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FOREWORD

Emory University School of Medicine takes pleasure in sponsoring this issue of *The American Surgeon*, which is devoted to work of the Joseph B. Whitehead Department of Surgery. Surgery has always occupied an important place in the history of this school and the name of Daniel Collier Elkin is especially prominent in its development. He was professor and chairman of the department from 1930 to 1954 and it was during this period that he stimulated and directed the first significant clinical research programs at Emory. Through his efforts and, in part due to his leadership, the Joseph B. Whitehead Chair of Surgery was established in 1939. Several years later further generous gifts made possible the endowment which was appropriately recognized by establishment of the Joseph B. Whitehead Department of Surgery. In a real sense, this issue of the American Surgeon honors not only Emory University School of Medicine but also the memory of Dr. Elkin.

ARTHUR P. RICHARDSON, M.D., *Dean*

CRANIAL VASCULAR LESIONS IN NEUROLOGIC SURGERY*

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A cerebral vascular problem is usually classified by most doctors as an apoplexy or a ruptured intracranial aneurysm. The real apoplectic patients probably should be the concern of the organic neurologist, even though there are many cranial vascular lesions amenable to neurologic surgery. These surgically remediable situations begin with certain scalp blood vessel developments, and can be located in any anatomical structure of the cranium and its tissue contents. In actuality, the surgical approach to the improvement or remedy even for the intracranial blood vessel derangements may be at cervical or upper mediastinal anatomical levels. Certain common and rarer surgical encounters will be illustrated and other surgical pathologies will be briefly mentioned, since it is not possible in this communication to discuss all of the vascular lesions from the authors' experience.

Arteriovenous Malformations of the Scalp

The uncommon development or arteriovenous malformations of the scalp may be of congenital or traumatic origin. Their significance to the patients is usually cosmetic or of a pulsile auditory character. Early woodcuts depict the enormous size which such scalp developments may attain. In the smaller lesions of the scalp in the newborn, recession and total disappearance may occur with the child's growth. The arterial contributors to the adult progressions are from the facial, temporal and occipital branches of the external carotid artery. The supraorbital vessels, branches of the internal carotid arteries, may be the primary blood providers for these cirsoid acquisitions. These lesions grow, they pulsate, have a palpable thrill and an audible bruit. We have not been able to demonstrate any influences on these patients' systemic blood pressures with the mechanical obliteration of even the largest of such experiences. Surgical destruction is the only treatment. Such technicalities are those of scalp reflections. These must extend a bit beyond deformities as calculated from the physical examination. The essentials are those of dissection, vessel ligations, and electrocoagulation until every blood vessel participant has been destroyed. One may, in such a necessary destruction, seriously compromise the scalp vascular supply. If such is evident, multiple skull trephining may just as well be done at the time of the primary surgery. These skull openings down to the dura will rapidly produce a healthy granulation tissue bed for subsequent skin grafting.

Hemangioma Skull

Figure 1 shows a transversely sectioned surgical specimen of hemangioma skull. These growths produce tumor deformities, usually of the convexities of the cranium. Cosmetic but not intracranial symptoms dominate, although local pain or nondescript headaches are frequently noted. X-ray diagnoses are commonly indicative of intracranial disease. Further, the insidious expansions are of great concern to the present day "tumor conscious" laity. Circumcisional removal, sacrificing approximately 1 cm. of the normal skull about the base of the tumor, assures against a recurrence. In spite of the radiologic appearance and the histologic classification, these growths are rather avascular. Surgery can be safely completed with an attending cranioplasty if proper care is taken not to open the underlying dura.

Arteriovenous Malformation of the Dural Vessels

Arteriovenous malformation of the dural vessels are of both traumatic and congenital origins, developing between the middle meningeal artery and the dural venous channels. In the traumatic group, the radiologic evidence will be that of a fracture line traversing a dural sinus. If this fracture traverses the petrosal bone, one will, on mento-vertex roentgenographic position, see an enlarged foramen spinosum. In the instance of a congenital development within this

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FIG. 1

same category, the severely hypertrophied grooving of the inner table of the skull by the middle meningeal artery will suggest the proper diagnosis. Angiography will outline a normal internal carotid system. An isolated contrast study of the external carotid vessel illustrating dura-artery malformation (fig. 2) in a victim of unexplained focal convulsions who suffered a spontaneous subarachnoid hemorrhage, led to the aforementioned investigations. The exclusion of these vascular anomalies, congenital or acquired, requires the obliteration of the arterial feeder, the arteriovenous pool, and all of the venous conduits. A simple ligation of the external carotid or the middle meningeal will be totally ineffective.

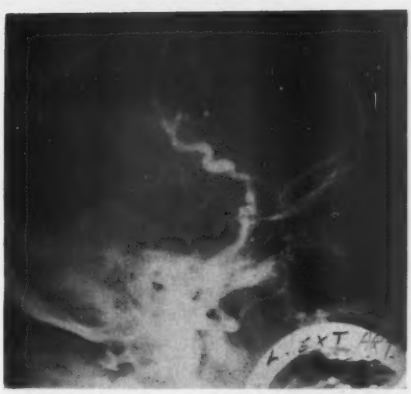

FIG. 2

Depressed Fractures in Which Larger Dural Sinuses Are Implicated

The neurosurgeon also encounters depressed fractures in which the larger dural sinuses are implicated. An illustration of involvement of the superior longitudinal sinus is seen in figure 3. This patient suffered an immediate paraplegia. The venogram depicts the compromise in the R. zone. The early elevation of the depressed skull probably played no important role in this patient's recovery of motor functions; however, credit for his rapid and complete restoration for ambulation was attributed to surgery.

Chronic Subdural Hematoma

One cannot discuss the cerebral vascular responsibilities of the neurosurgeon without commenting on the rather common occurrence of chronic subdural hematoma. These minor or intense situations appear in one's medical practice or in the occupancies of hospitals as acute and sometimes difficult cerebral diagnostic problems. The medical histories are fallacious, the neurologic objective evidence is variable, and all simple diagnostic studies may be either accurate or

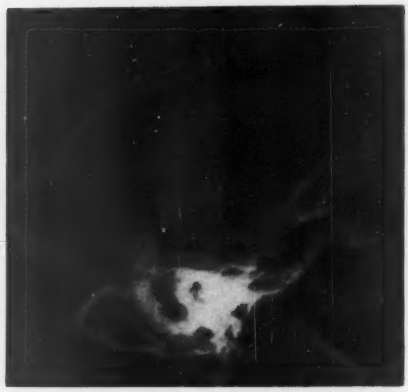

FIG. 3

entirely valueless. A situation "from the cradle to the grave," and it is in this group of surgically treated patients that the neurosurgeon should have his highest "batting average." In almost 30 years and over 300 personally operated cases, there is a surgical mortality of less than 1 per cent and no morbidities attributable to these patients' "chronic pachymeningitis hemorrhagica." It is the authors' opinion that these low figures are attributed to the following routines: (1) local anesthesia; (2) bifrontal trephines, perhaps a bit more laterally placed in such suspects than for routine ventriculography and trephination performed in face-down (cerebellar) positions; and (3) ventriculography, if no subdural pathology is encountered. There is no excuse for multiple trephines all over the skull. Dr. Walter Dandy made one of the greatest contributions to neurosurgery when he injected air into the cerebral cavities and followed such with skull roentgenograms. Thus, there is no necessity for so-called "pecker-wood" skull surgery. Gravity will do more for bulk cerebral expansion than intraspinal or intraventricular fluid injections. Lowering the head below body level following the subdural evacuation of the hematoma, awaiting time for cerebral expansion and the appearance of normal cerebral pulsations before wound closure, permits a reliable prognostication.

"Vascular Lesions Suspects" Presenting Differential Diagnostic Problems

The extensiveness of one's experiences always nets a few situations in which the neurologic facts may be proved "not what they seem." Such an experience was that of a middle-aged man who had an extensive scalp-face-neck telangiectasia from birth. This alarming "birthmark" was ipsolateral to the side of his adult developments of cerebellar pontine symptoms. Serious permanent residuals, including blindness, were anticipated preoperatively. A benign acoustic neuroma was completely extirpated, even though this patient had been assured over a period of 5 years and by double that number of capable diagnosticians that his intracranial handicaps were on the basis of disease comparable to his face-head blemishes. Another illustration of preoperative diagnostic error is shown in figures 4 and 5. The calcific outline was that of an enlarged intracranial carotid artery. It was thought, because of the abruptness of the onset

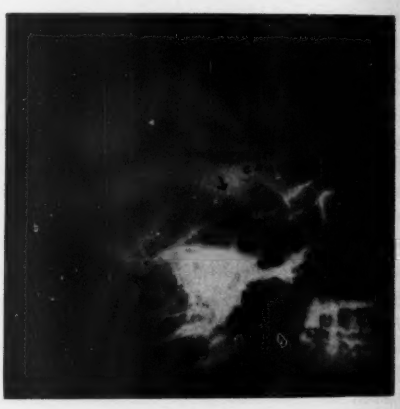

FIG. 4

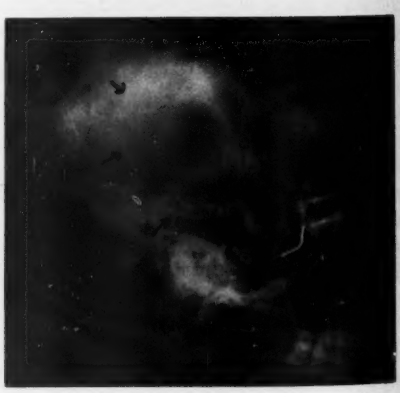

FIG. 5

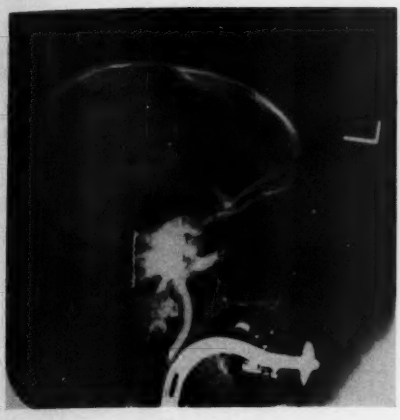

FIG. 6

of symptoms, that an intracerebral hemorrhage was the proper diagnosis. At the time of the craniotomy for the evacuation of this woman's cerebral occupant, the ironing-out of the cerebral branches of the carotid was found to be the result of a large intrinsic "neuroblastic glioma." She remains well 18 years since her initial diagnosis of an intracerebral artery lesion.

Cavernous Sinus Fistulas

Cavernous sinus fistulas may appear spontaneously or may be initiated by a trivial trauma. The pulsating unilateral exophthalmus is the more common presentation. Carotid angiography outlines a "down draft" of the injected contrast media at subclinoidal levels. The visualization of the intracerebral divisions of this vessel is usually not accomplished. Figure 6 is an illustration of one of several attempts to see the intracerebral arteries. These situations may be eradicated by a simple cervical ligation. Occasionally, a craniotomy and carotid ligation at clinoidal level is necessary. If such has to be performed, one should dissect out the ophthalmic artery at the time of the craniotomy. The ophthalmic artery should then be doubly ligated and severed. Such will prevent a secondary craniotomy for the exclusion of the perseverance of the ocular symptoms and an audible bruit.

Intracranial Aneurysms

The surgical attack on intracranial aneurysms, these arterial out-pouchings, seems to be a "fielder's choice." The ideal accomplishment, of course, is the total elimination of such an acquisition. In figure 7 is shown one of our earliest experiences in cerebral angiography, performed in the operating room as an "open injection" using 8 cc. of Thorotrast. Previously, I had discussed the surgical possibilities of intracranial aneurysms with the late Dr. Walter Dandy. I misunderstood him, for I thought he told me that he had cured such a patient by "trapping the aneurysm." When I talked to him later and told him that his procedure had been effective in this patient's behalf, he said, "I did not say I had so operated but I told you a cure could be expected from such a procedure." He was correct in his postulation. This patient (fig. 7), but for a residual VIth nerve palsy, remains well and successfully self-employed now 18 years since his surgery. Figure 8 is the artist's depiction of "the trapping procedure." There occurs at times in younger adults a spontaneous unilateral ophthalmoplegia externa. These may develop insidiously without a history or any evidence of intracranial bleeding. These congenital lesions may likewise manifest themselves as the explanation for a unilateral loss of vision. A "stalk" of a millimeter or so may be available for such a surgical aneurysmal detachment. This may be accomplished with the use of the silver clip or a heavy silk ligature. Figures 9 and 10 are illustrative of such experiences. For security, "the stalk" in this one instance was doubly clipped.

Table 1 gives the over-all surgical and autopsy figures of the intracranial aneurysmal sites. It is noteworthy that more than 85 per cent of all intracranial aneurysms are surgically available. There are a few minor aneurysms from the

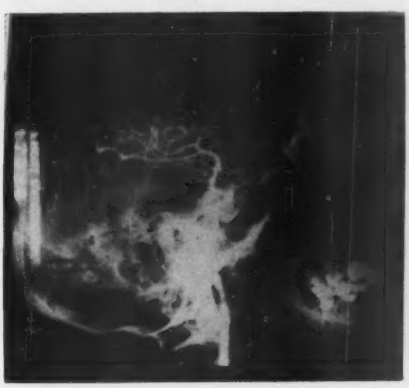

FIG. 7

vertebral, as well as the basilar, artery branches which can rupture and manifest themselves as the etiology of a spontaneous subarachnoid hemorrhage. Further, some such lesions, without bleeding, may manifest themselves as cerebellar pontine angle space occupants or irritants. Certain divisions of the major cerebellar arteries may be the locales of congenital aneurysms.

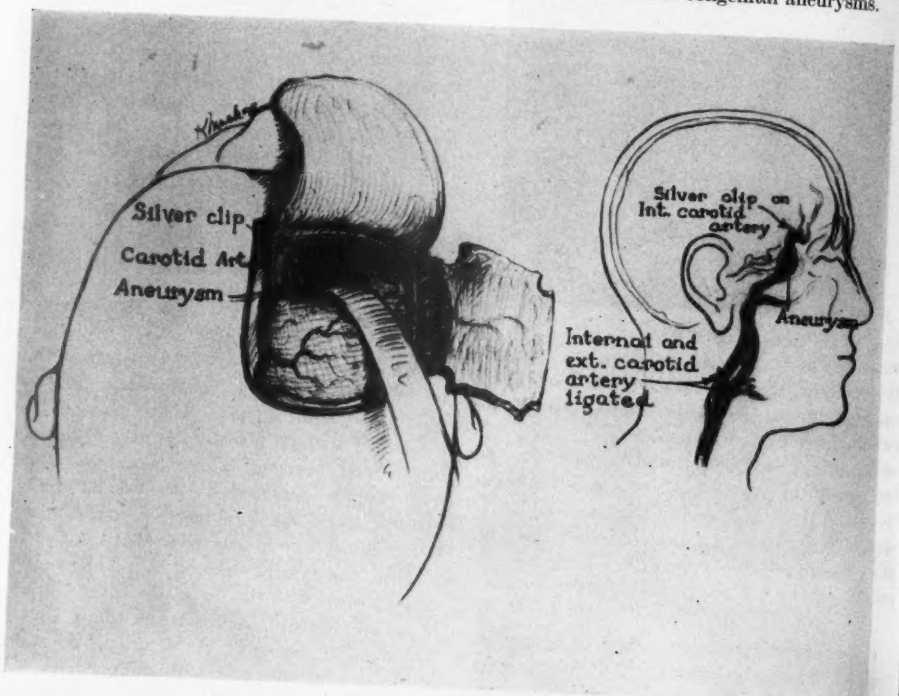

FIG. 8

FIG. 9

FIG. 10

TABLE 1

Locations of intracranial aneurysms

	Per Cent
1. Within carotid canal	10
2. Intracranial carotid	35
3. Anterior cerebral	10
4. Anterior communicating	10
5. Middle cerebral	20
6. Posterior communicating	1
7. Posterior cerebral	2
8. Basilar and vertebral	12

Intracranial Arteriovenous Malformations

Intracranial arteriovenous malformations are congenital maldevelopments. Although more common in the cerebrum, the cerebellum can be the site for such a blood vessel mass. These anomalies may manifest themselves in an irritative or hemorrhagic manner. The angiogram reproduced in figure 11 was one of intracranial hemorrhage in a child. Further explanation for the repeated Jacksonian convulsions, which antedated the bleeding, was established on the initial review of this arterial visualization. Angiographic follow-up study of this child depicted the surgical exclusion of the arterial anomaly and the over-all physical improvement of the cerebral circulation. Figure 12 shows an adult malformation. As is seen, a craniotomy had preceded this angiogram. The operating surgeon felt his experience in this instance would not permit a safe attack. This is correct. One should, in these craniotomies, assume a philosophy of "all or none." The primary attack must be the obliteration of all the arterial contributors. Once this has been accomplished, the extirpation of the venous components is a relatively easy performance. Figure 13 shows a postoperative study of figure 12.

Mass Sized Intracranial Aneurysms

There are a few cranial occupants of blood vessel origin which, clinically, masquerade as "brain tumor suspects." X-ray films may complement such a diagnosis. Such an experience is illustrated in figure 14. The artist's composite sketch, made from this patient's anterior-posterior ventriculogram, is shown in figure 15. The exclusion of the mass occupancy is imperative for the relief of the intracranial symptoms in these persons. Ligation of the stalk and preservation

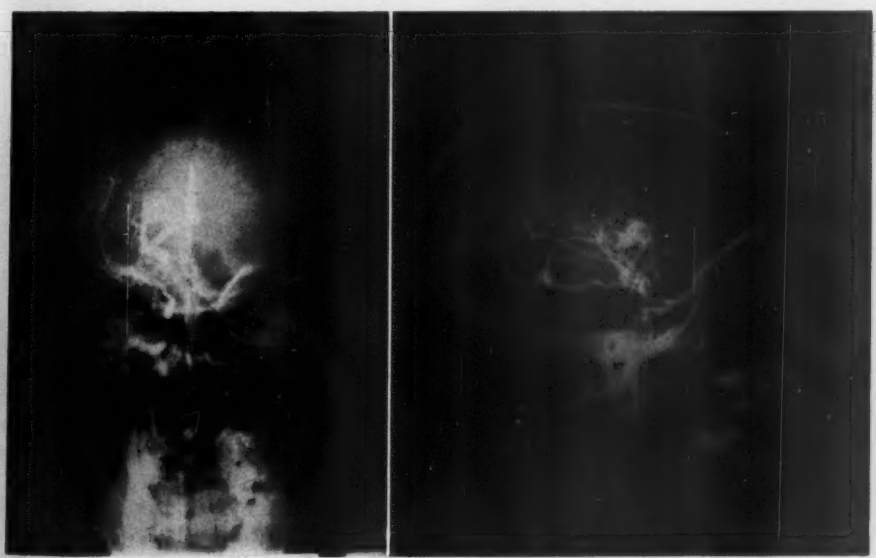

FIG. 11

of the normal circulation will leave the patient with no surgically acquired neurologic handicaps.

Intrinsic Hematoma Produced by Mycotic Cavernous Angioma

The incidence of a hemorrhage into a pre-existing brain tumor is such a rare event that it is hardly worthy of mention. The incidence of a spontaneous hematoma within the cerebral or cerebellar substances is more common. The miniature cavernous angioma shown in figure 16 was accidently salvaged from a small sized suction tip in the course of evacuation of a large temporal lobe organized blood clot. The diameter of this gross lesion occupied the middle one-third of the conventional cover glass used in routine microscopic tissue mountings.

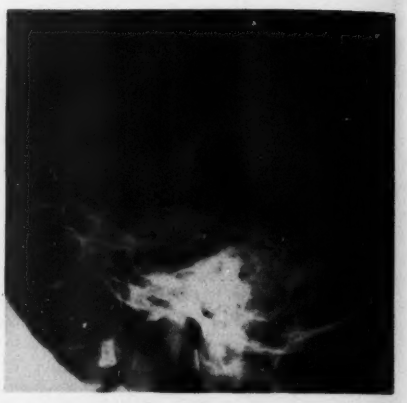

FIG. 14

FIG. 12

FIG. 15

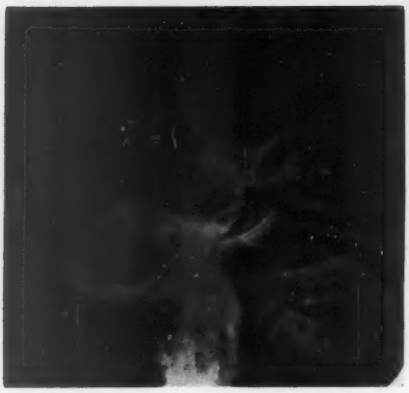

FIG. 13

Blood Vessel Inadequacies

It is not known whether blood vessel inadequacies are developmental or there is some irritative situation which causes a reduction in blood vessel calibers. Periodic, alarming symptoms are to be found in patients suffering from these rarities. These are not the arteriosclerotic victims nor is the insufficiency demonstrable as cervical carotid disease. Focal convulsions, attended by recurrent hemiplegias, were encountered in the patient (fig. 17). In this

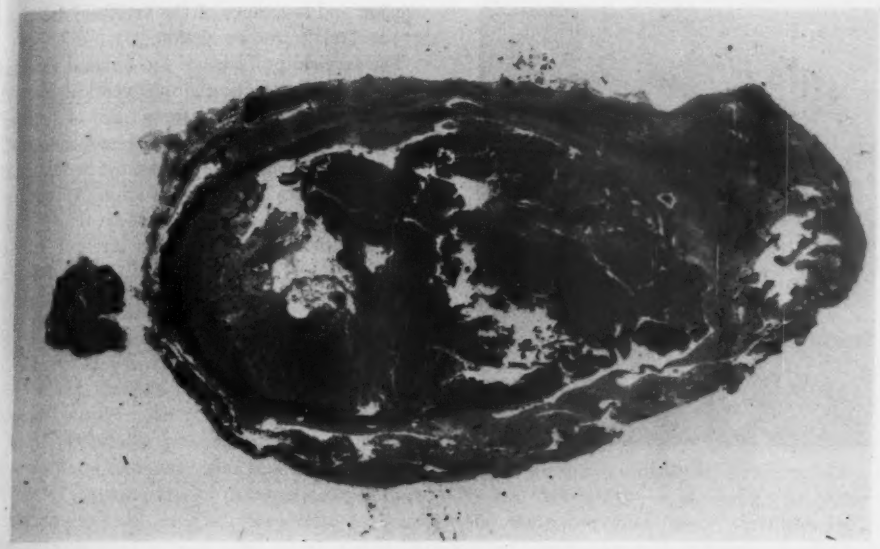

FIG. 16

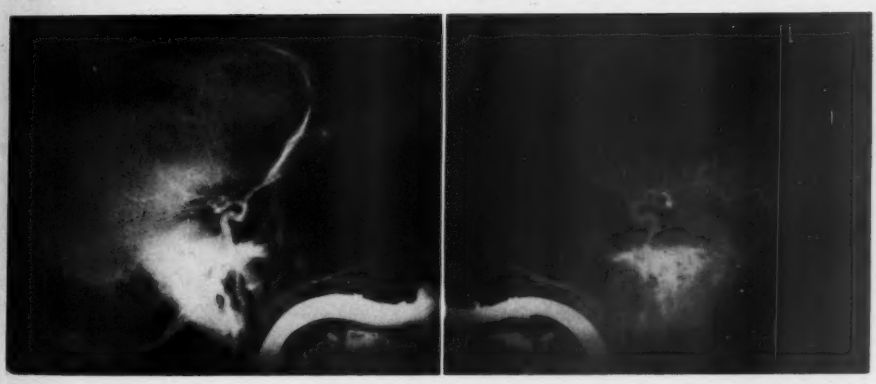

FIG. 17

instance, repeat angiograms failed to depict the caliber of vessels as outlined on the opposite side. In some of the authors' experiences, sympathectomy has been of apparent benefit.

Cervical Vertebral Lesions

Figure 18 is illustrative of the spontaneous development of cervical vertebral lesions. Some of these "noisy situations" may be dispensed with by performing a simple ligation of the vertebral artery at its parent origin. Other surgical efforts may require intraspinal severance of this artery. This would be evaluated as another "trapping operative procedure." Further efforts for the exclusion of such a vertebral aneurysmal bruit may involve one in an ipsolateral or bilateral external carotid exclusion. Such a surgical venture may end in a multiplicity of operative efforts, and the "arterial noise" may continue. In the instance so illustrated, a complete disappearance of all subjective and objective evidences of the vascular insultant followed a

FIG. 18

ligation and severance of the vertebral artery at its subclavian artery emission.

The surgical problems of the cerebral vascular insufficiencies resulting from cervical carotid vascular obliterative disease are of current interest and efforts by all surgeons concerned. The evaluation of these efforts must be tabulated with long term follow-up studies. Apropos of these surgical ventures, it must be remembered that a patient deprived of his cerebral arterial needs for a reasonable period of time is doomed. With irreversible cellular brain damage there is permanent cerebral morbidity. The surgical help for these patients must antedate total arterial occlusion.

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THE USE OF THE T-TUBE IN DIFFICULT DUODENAL STUMP CLOSURES*

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Management of the difficult duodenal stump is frequently a major problem in gastric resection for duodenal ulcer. This is most likely to present itself in cases where emergency resection is performed for acute massive hemorrhage and where the bleeding ulcer is located on the posterior duodenal wall and is penetrating.

Where extensive periduodenitis is encountered and where massive hemorrhage is not the immediate problem, any of the exclusion procedures can be performed, providing the antral mucosa is eventually removed. Dire consequences manifested by pancreatitis and pancreatic fistula have resulted from ill advised dissection of some posterior ulcers from the head of the pancreas. When there is foreshortening of the duodenum, extensive inflammatory reaction, or adherence of the ulcer to the common duct, identification of the common duct is important. Lahey's method of introducing a T-tube in the duct will minimize possible damage to this structure.[3]

Occasionally, the surgeon finds that he has proceeded to a stage where exclusion is not feasible and the ulcer has to be exteriorized. The essential feature here is that the ulcer is *not* dissected from the head of the pancreas but is excised from the duodenal wall and thus separated from the continuity of the duodenal lumen. Inversion of the duodenal stump may be accomplished by a plastic procedure as described by Graham,[2] Nissen,[4] or Bsteh.[1]

In cases of massive hemorrhage with marked periduodenitis and where a bleeding vessel in a posterior indurated ulcer bed must be controlled, the problem of closure of the duodenal stump is magnified. This requires exposure of the ulcer bed and the situation arises where the remaining inflamed duodenal wall is inadequate for a secure closure and further dissection would endanger the pancreatic or common bile duct. Catheter duodenostomy through the stump as described by Welch[5] affords an excellent solution to this difficult and hazardous situation. In an effort to diminish morbidity and complications, a modification of Welch's catheter duodenostomy was suggested by Dr. J. C. Thoroughman, Chief of Surgery, of this hospital. In place of a catheter through the stump or open end of the duodenum, a T-tube is inserted into the second portion of the duodenum for decompression and as a controlled duodenostomy in those cases where it is evident that a secure inversion of the stump is improbable. This serves as a protective measure against the grave sequelae of duodenal blow-out. We feel that it is more physiologic to bring the tube through normal tissue than through the scarred, friable, edematous tissue present in this type of duodenal stump; the fistulous tract heals more rapidly and there is less leakage of biliary and pancreatic juice about the tube.

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TECHNIQUE

The technique used at this hospital is as follows. Exploration is made through a midline incision and if the hemorrhage is of such magnitude as to require immediate control a gastrotomy is made. This is carried through the pylorus into the duodenum, the ulcer bed is visualized and the bleeding vessel is controlled temporarily by finger tamponade or stitch ligature. Occasionally, the condition of the patient precludes a resection and ligature control of the bleeding vessel may have to suffice. The gastroduodenotomy can be converted into a pyloroplasty and a vagotomy performed, followed by T-tube decompression of the duodenum if the pyloroplasty is insecure.

If the condition of the patient permits, the gastric resection is carried out in the usual manner when the hemorrhage is controlled or if there is evidence of temporary cessation of bleeding. When marked periduodenitis is present there is no hesitation in opening or dividing the duodenum proximal to the ulcer and inserting the finger into the lumen for better identification and visualization. Frequently one enters the duodenum accidentally at the superior margin of the ulcer during the periduodenal dissection. The

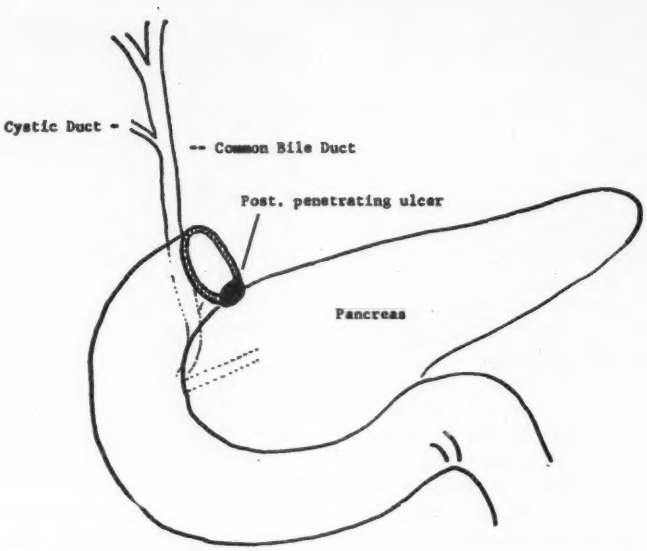

FIG. 1. The duodenum has been divided at the level of a large posterior penetrating ulcer. Note the close proximity of the ulcer to the common bile duct. The ulcer is separated from the duodenum by cutting along the indurated edge of the ulcer. This exteriorizes and leaves the ulcer base on the pancreas.

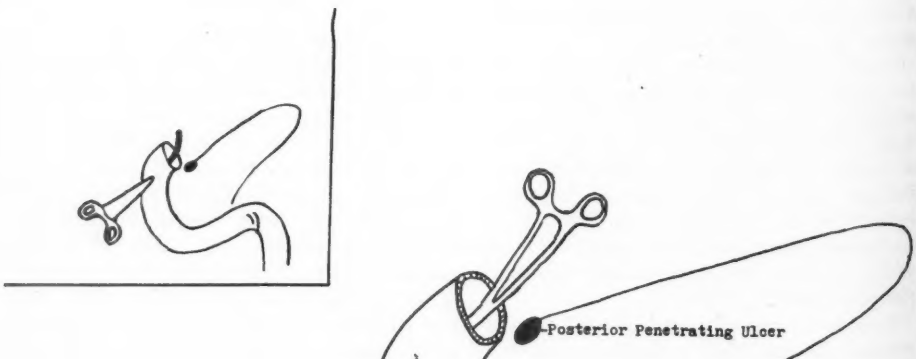

FIG. 2. A curved hemostat is inserted through the open end of the duodenum and a puncture wound made approximately 1½ inches distal to the stump. The short arms of the T-tube are pulled into the lumen under direct vision. An alternate method is shown in the inset.

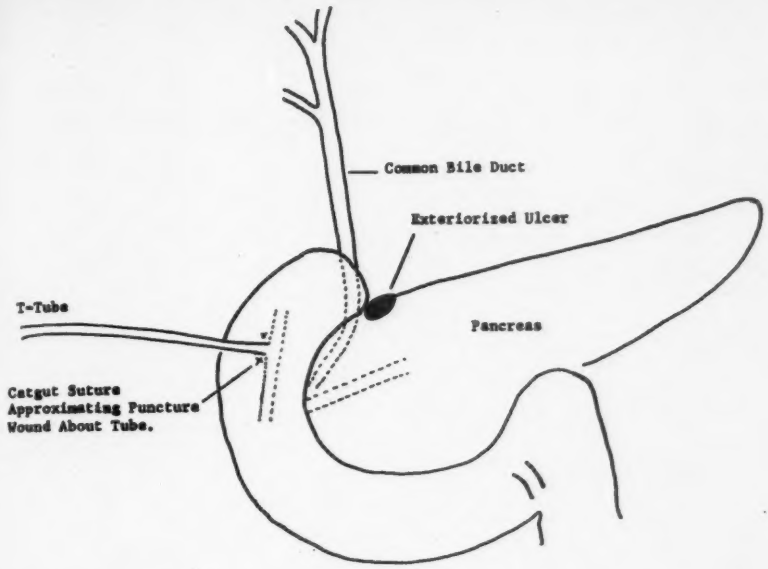

FIG. 3. The T-tube has been inserted, the duodenal stump inverted, and the ulcer exteriorized

ulcer is freed from the duodenum by cutting along the entire scarred ulcer circumference, being careful to exclude mucosa and to avoid deep penetration beyond the serosa. This exteriorizes and isolates the ulcer bed from continuity of the duodenum and leaves the base on the pancreas (figs. 1 and 3). When the ulcer is freed from the duodenum there is an increase of mobility, allowing more duodenal tissue for closure. No clamp is placed on the distal duodenum so as to preserve all available tissues for inversion or closure of the stump. Constant awareness of the location of the common duct is emphasized. The majority of these ulcers are active, posterior, penetrating and are in close proximity to the common duct.

If it is evident that inversion of the duodenal stump will be unsatisfactory, a T-tube is utilized (figs. 2 and 3). With the aid of a right angle or curved clamp, a puncture wound is made through normal duodenal tissue in the second portion of the duodenum approximately 1½ inches distal to the stump. This can be done before or after closure of the stump, although it is easier to perform this maneuver before closure. The short arms of the T-tube are pulled into the lumen of the duodenum easily under direct vision. The proximal arm of the T-tube is cut short so that it is not caught in the inverted stump. If the decision is made to use a controlled duodenostomy after the stump is closed, the T-tube can be introduced in the same manner as one inserts a T-tube in the common duct. One or two catgut sutures are used to approximate the puncture wound tightly about the long arm of the T-tube as it emerges from the duodenal wall. A 50 per cent gastrectomy, subdiaphragmatic vagotomy and gastrojejunostomy complete the procedure. A Penrose drain in the subhepatic area and the long arm of the T-tube are then brought out through a stab wound in the upper right quadrant. Retention wire sutures are used in cases where emergency resection for massive hemorrhage is performed. The long arm of the T-tube is usually connected to gravity drainage.

DATA AND DISCUSSION

Tables 1 and 2. From 1955 through 1958 the use of the controlled duodenostomy has been employed 14 times (6 per cent) in 243 resections. Of these resections, 39 were emergency procedures for massive hemorrhage and in 11 of these (28

TABLE 1

Type and number of controlled duodenostomies in 243 gastric resections

Year	Gastric Resections	T-Tube	French Catheter (Welch's Method)	Total
1955	48	0	1	1
1956	61	2	0	2
1957	57	1	0	1
1958	77	8	2	10
Total...	243	11	3	14

TABLE 2

Year	Emergency Resections	Controlled Duodenostomy
1955	9	0
1956	8	2
1957	6	0
1958	16	9
Total.....	39	11

TABLE 3

Complications (in 16 cases)

A. None (uneventful postoperative course)..	7
B. Minor	
1. Small SQ abscess at T-tube site*.....	1
2. RUQ and back pain when T-tube clamped*.....	1
C. Major	
1. Local chemical peritonitis.....	3
2. Jaundice.....	2
3. Wound disruption.....	1
4. Fecal fistula and peritonitis.....	1

* Complications related to duodenostomy.

per cent) controlled duodenostomy was employed. Controlled duodenostomy was utilized 3 times (1.4 per cent) in 204 elective resections. During 1958, 16 emergency procedures were done for acute massive hemorrhage. Of these, 9 (56 per cent) had controlled duodenotomies, illustrating the increase in frequency of its use during the past year. So far, in 1959 (January through March) we have performed 2 additional T-tube duodenostomies in 14 elective resections. This brings the total to 16.

Table 3. The complications associated with these difficult closures requiring duodenostomy are shown in table 3. No deaths occurred; 7 had uneventful postoperative courses. There were 2 who had minor complications. Right upper quadrant and back pain were noted in 1 patient when the T-tube was clamped for 24 hours. Pain was relieved when the T-tube was unclamped. A small subcutaneous abscess in the T-tube tract constituted the other minor complication.

Local chemical peritonitis was noted in 3 patients. Of these, 1 had stoma obstruction and the remaining 2 had clinical findings suggestive of duodenal leakage. The T-tube decompression obviously offered great insurance against the sequelae of sudden duodenal blow-out in these cases with insecure stump closures. It was supplanted by a mild to moderate local chemical peritonitis manifested by fever, right upper quadrant pain, and ileus of several days' duration. Symptoms subsided when adequate drainage of biliary material appeared on the dressing *via* the drains in the upper right quadrant. One is led to conclude that bilary and pancreatic juice leaked around the tube or through the duodenal stump. We feel that a catastrophe was avoided in these 3 cases by the use of the T-tube.

Obstructive jaundice occurred in 2 patients, the bilirubin rising as high as 2.9 and 9 mg. per cent by the fifth and sixth postoperative day and gradually returning to normal levels by 14 to 19 days. In one case no bile was noted in the T-tube for 4 days; however, copious amounts flowed through and around the tube on subsequent days. It was felt that these 2 cases gave further support for the judicious use of the controlled duodenostomy and decompression in preventing or minimizing duodenal leakage with its associated complications. The complications of wound disruption and fecal fistula were unrelated to the use of the T-tube.

The Penrose drain is usually removed on or about the 10th to 12th postoperative day and the T-tube, about the 20th day after it had been

TABLE 4

Mortality (1951 through 1958)

	Gastric Resections	Deaths	Per Cent
Emergency.....	63	4	6.4
Elective.....	358	4	1.1
Total.....	421	8	1.9

clamped for 5 to 7 days without untoward symptoms. Following removal of the T-tube a small drain is inserted into the tract for a distance of 1 to 2 inches for several days to prevent the skin from closing over the tract too quickly with subsequent abscess formation, as occurred in 1 of the cases. The fistulous tract closes in 1 to 3 days.

The average daily T-tube drainage is approximately 400 to 800 cc. The greatest amount of drainage is noted (up to 2500 cc.) in those cases with stoma obstruction. The amount of drainage usually increases by 300 to 1000 cc. on the fourth or fifth postoperative day and this coincides with the onset of oral feedings. The sudden increase is probably due to the passage of oral fluids through the gastrojejunostomy stoma into the afferent loop and illustrates the advantages of the decompression afforded by the duodenostomy tube. In 4 cases the maximal daily drainage was less then 500 cc. and in 2 cases, less than 250 cc.

Table 4. From 1951 through 1958 there have been 421 gastric resections with a mortality of 1.9 per cent. For elective cases the mortality has been 1.1 per cent and for emergencies 6.4 per cent. During this period there have been 10 duodenal leaks or fistulas with 2 deaths attributed to the fistulas. Of these fistulas, 7 followed gastric resections and 4 of the 7 occurred after emergency resections for massive hemorrhage; 3 followed duodenotomies, 1 of which died. Of the 10 cases with fistulas, 8 had Penrose drains inserted at the time of the original operation. Of these, 6 had stormy postoperative courses and 1 died, supporting the impression that Penrose drains give incomplete protection in cases of duodenal leakage or blow-out. Analysis of the 8 deaths following gastric resection strongly indicates that use of the T-tube probably would have prevented the demise of 2 of the 8. We also feel that the death following the duodenotomy probably would have been prevented by the use of a controlled T-tube duodenostomy.

In 1958, during which controlled duodenostomy was used frequently, there were no fistulas in 16 emergency and 61 elective resections. The 1 death in 1958 was due to postoperative hemorrhage.

SUMMARY

We have used T-tube drainage as a controlled duodenostomy and decompression of the duodenum in 16 cases where the integrity of the closure of the stump was in doubt and we feel that it seems more physiologic to bring the tube out through normal duodenal wall rather than at the end of the stump where the tissue is inflamed, friable and edematous. The morbidity has been found to be minimal and no fistula of consequence has occurred in this small series. The use of the T-tube has increased in incidence over the past year, and we now use it whenever there is doubt about the integrity of the closure. Clinically, one is most apt to employ the controlled duodenostomy in those cases requiring emergency gastric resection for acute massive hemorrhage and where the ulcer is active, posterior and penetrating. It is a simple procedure that can be performed quickly and has proved to be safe when indicated. It is superior and affords more protection than Penrose drainage alone.

The principle of controlled duodenostomy can be applied to traumatic perforation and surgical duodenotomy as well as to the duodenal stump whenever the closure is insecure or in doubt. Review of our records indicates that three deaths resulting from gastroduodenal surgery may have been prevented by its use.

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REFERENCES

1. Bsteh, O.: Technik der Resektion tiefsitzender Duodenalulcera. Arch. klin. Chir., *175:* 114, 1933.
2. Graham, R. R.: Surgical therapy in lesions of the stomach and duodenum. In *Operative Surgery*, edited by F. W. Bancroft. Appleton-Century-Crofts, Inc., New York, 1941.
3. Lahey, F. H., and Marshall, S. F.: The surgical management of some of the more complicated problems of peptic ulcer. Surg. Gynec. & Obst., *76:* 641, 1943.
4. Nissen, R.: Zur Resektion des tiefstzenden Duodenalgeschwürs. Zentralbl. Chir., *60:* 483, 1933.
5. Welch, C. E.: Treatment of acute, massive gastroduodenal hemorrhage. J. A. M. A. *141:* 1113, 1949.

TRANSAXILLARY TRANSPLEURAL SYMPATHECTOMY FOR THE UPPER EXTREMITY*

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Transaxillary transpleural sympathetic ganglionectomy is a relatively new approach to performing a sympathectomy for the upper extremity. It was originally devised by W. G. Schulze and Professor R. H. Goetz (of Capetown, South Africa). In theory, it is believed to offer the most complete and most permanent method. In actual practice, preliminary observations confirm these beliefs. The transaxillary transpleural sympathectomy is performed by entering the pleural space through the second or third interspace and then excising the inferior half of the stellate ganglion, the 2nd, 3rd and 4th ganglia and intervening chain.

Although the popularized preganglionic nerve root section and section of the sympathetic trunk was advocated by Telford[20] in 1935, and Smithwick[19] in 1936, to prevent regeneration of nerve fibers and to decrease vascular spasm from Adrenalin sensitization, the reasons for instituting this method have not been validated. In fact, there is considerable evidence that it does not accomplish its purpose. A few early clinical observations had indicated that patients having ganglionectomies developed recurrent vasospasm in the upper extremity within short periods of time (from 10 days to 1 year). The convincing preliminary evidence for inaugurating "preganglionic" sympathectomy was primarily based on animal experiments.[2, 7, 18] It was suggested that cervicothoracic ganglionectomies were different physiologically from lumbar ganglionectomies, which give good and lasting results for the lower extremities, in that removal of L1 and L2 ganglia in reality constitutes a preganglionic sympathectomy for the lower extremity and does not cause death of the terminal sympathetic nerve fibers going to the lower extremity. The cell bodies for these neurons have been demonstrated to lie in the ganglia at L3, through S3. Even inclusion of L3 in the ganglionectomy was thought to produce degeneration of such a small number of nerve fibers that its effect in producing Adrenalin sensitivity is negligible.

A large amount of additional evidence has now accumulated to demonstrate that the evidence acquired from the animal experiments, indicating sensitization to Adrenalin after ganglionectomy, cannot be applied to humans.

Fatherree and associates,[4] after many carefully controlled clinical experiments, disagreed with the clinical observations and conclusions of Smithwick and associates.[18] They demonstrated that the vasoconstrictor effect of epinephrine varies widely in normal individuals and also is not necessarily exaggerated in patients with Raynaud's disease. They demonstrated that sympathectomy of the upper extremity by removal of the ganglia does not produce nearly as great sensitization of smooth muscle in man as it does in monkeys. In some patients, both preganglionic sympathectomy and ganglionectomies for the hand effected a decrease in cutaneous temperatures in response to standard doses of epinephrine that was twice that preoperatively.

Hyndman and Wolkin[11] compared results of the standard preganglionic sympathectomy done on one side for the upper extremity with ganglionectomy on the other in the same patient. No difference in sensitivity to Adrenalin could be demonstrated after sufficient time for nerve degeneration had elapsed.

Simmons and Sheehan,[17] in reviewing the preganglionic sympathectomies and the ganglionectomies done for the upper extremity in Telford's Clinic, found very little difference in the early and late relapses following each of the two types of procedures. Their observations led them to conclude that early relapses were due to local faults of the vessels and that the late relapses may be due to transient hypersensitivity to Adrenalin.

There has been much question as to whether the "preganglionic" sympathectomy of Telford and Smithwick is truly preganglionic. Adson[1] suggested that during the operation and manipu-

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lation around the ganglia the blood supply may be damaged, causing death to the ganglion cells. Grimson[9] suggested the same possibility and also pointed out that division of the chain at T3-T4 or T4-T5 may actually be a partial postganglionic operation, since synapsis may occur lower down the chain. Demonstration of the location of the terminal ganglia bears him out.

There is evidence that the preganglionic nerve cells supplying the upper extremity arise in the human as far inferiorly as T10. Ray and associates,[16] during laminectomies, made observations in 7 patients to determine the lowest level of supply to the hand. Faradic stimulation of the various nerve roots was accomplished and changes in electrical resistance of the skin were measured. The lowest levels were found to vary between T7 and T10. In 11 other patients these investigators also determined the highest preganglionic outflow to the upper extremity to be between T1 and T3. The usual place of greatest outflow was midway between the uppermost and lowest point of innervation. Hyndman and associates[12] showed that the preganglionic fibers for the upper extremity originate in the spinal cord segments from T4 to T8.

The weight of evidence indicates that nerve root section of T2 and T3 or of T2, T3 and T4 would only interrupt a very small proportion of the preganglionic fibers going to the upper extremity, and that if this method were to be used, all nerve roots from T2 through T10 would have to be sectioned. This would be impracticable because it would give an undesirably wide zone of anesthesia of the chest wall and paralysis of muscles of the intercostal spaces.

There is considerable evidence to show that regeneration of sympathetic nerves rather than hypersensitivity to Adrenalin plays an important part in the recurrence of angiospasm after sympathectomy. Simmons and Sheehan,[17] in re-evaluating patients who had pre- or postganglionic sympathectomies, found that in all cases of late relapse regeneration of constrictor fibers could be demonstrated by producing vasodilation after anesthetizing the median and ulnar nerves. In 1929, Lee[15] demonstrated regeneration of the cervico-sympathetic chain 275 days after the sternomastoid muscles had been placed between the divided ends of the chain. Other authors[8, 10, 13] have shown similar regeneration in experimental animals.

Numerous investigators within the past few years[3, 5, 9, 11] have stated that they prefer the clinical result of ganglionectomy to nerve root section and sympathetic trunk section. They have usually recommended removal of the 2nd, or 2nd and 3rd ganglia.

There are several objections to ganglionectomy through the cervical (anterior) approach. Removal of the stellate (inferior cervical and 1st dorsal) ganglion and sometimes the 2nd dorsal ganglion is accompanied by the production of the objectionable Horner's syndrome. It may be difficult to expose and remove the 2nd thoracic ganglion. Visualization and transsection of the nerves of Kuntz[14] are also seldom possible through the anterior cervical approach. In several of Hyndman and Wolkin's[11] cases the patients developed burning pain in the upper extremity following cervicodorsal sympathectomy. Adson[1] reported similar observations.

The posterior approach for ganglionectomy is an operation of considerable extent, which throws a great deal of strain on the patient's vitality. It involves rib resection and still does not provide good exposure for adequate ganglionectomy.

Goetz and Marr[5] have described an anterior approach for transpleural upper dorsal sympathectomy for the upper extremity in which an incision 4 inches long is made over the first or second intercostal space, commencing at the margin of the sternum. The pectoralis major is split in the direction of the fibers and the pleura is opened. The internal mammary artery is divided and adjacent costal cartilages are cut. The ribs are spread, and the 2nd thoracic ganglion is removed.

The transaxillary, transpleural sympathectomy is the simplest of all the procedures in use. It provides excellent anatomical exposure for removal of as much of the sympathetic supply to the upper extremity as may be needed.

TECHNIQUE

With an endotracheal tube in place, under nonexplosive anesthesia, the patient is placed in the lateral chest position with the forearm secured to the ether screen. A transverse incision over the second or third interspace is made from the lateral border of the pectoralis major to the lateral border of the latissimus dorsi (fig. 2). The long thoracic nerve which will be found in the prethoracic areolar tissue is identified, preserved and retracted from the operative field. The incision is carried down through the underlying

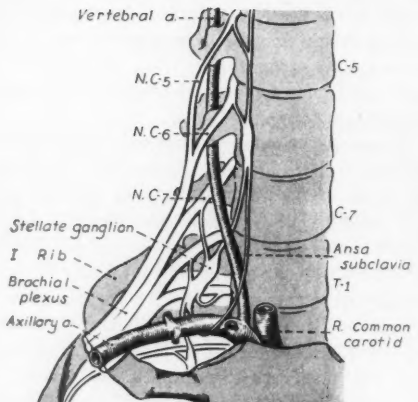

FIG. 1. Anatomy of cervical and thoracic sympathetic nervous system.

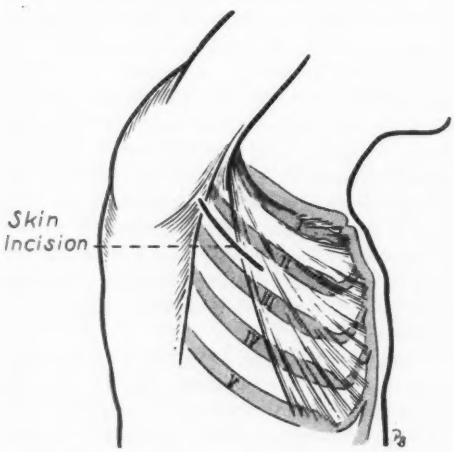

FIG. 2. Operative incision for transaxillary sympathectomy.

interspace and the pleura is opened. The small rib spreader is inserted into the interspace and the ribs are slowly spread. Only enough space for instrument operation is obtained. Any adhesions to the apical portion of the lung are divided. Minor bleeding is controlled by coagulation, silver clips or sponge pressure. The apex of the lung is allowed to collapse or is retracted to reveal the upper dorsal and stellate ganglia. The pleura is incised over the ganglia (fig. 3).

FIG. 3. Operative exposure of sympathetic nervous system through a transaxillary approach.

The rami communicantes to T2, T3 and T4 and the chain between T4 and T5 are compressed with silver clips and transected. The rami communicantes from T1 are left intact, since transection of them will produce a Horner's syndrome.[6] The stellate ganglion will still have a large portion that may be removed below the points of entry of these rami. This portion of the first dorsal ganglion is removed by placing multiple silver clips across the ganglion and transection inferior to the clips. The lung is reexpanded as the chest is closed. If it has been necessary to divide many adhesions to free the lung, catheter drainage to a water seal bottle is used.

COMMENT

The silver clips are used for control of bleeding and are firmly compressed on the nerve fibers to add this effect to minimizing regeneration. Attention is directed to anomalous rami or the nerves of Kuntz that may be present[14] (fig. 1).

Using the technique described above, 22 patients with arterial insufficiency of the upper extremity have been operated upon by the authors. Immediate and short term follow-up observations indicated excellent and complete sympathetic block of the upper extremity. Of these cases, 2 were reoperations for arterial insufficiency of Buerger's disease, which was recurrent after a dorsal "preganglionic" sympathectomy. In these cases also, excellent results were obtained.

SUMMARY

1. Transaxillary transpleural sympathectomy, removing the inferior portion of the stellate ganglion and the second, third and fourth ganglia, is the simplest and most complete method of accomplishing a sympathetic denervation of the upper extremity.

2. By preserving the rami communicantes of the first thoracic nerve and the superior portion of the stellate ganglion, Horner's syndrome was prevented.

3. For completeness of sympathectomy, the auxillary nerves of Kuntz, which may arise from the second thoracic nerve, the second thoracic ganglion and the third thoracic nerve, must be searched for and divided when present.

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REFERENCES

1. Adson, A. W.: Indications for and operative procedures on the sympathetic nervous system. In Lewis' *Practice of Surgery*, Vol. 3, Ch. 8, p. 1. W. F. Prior Company, Inc., Hagerstown, Md., 1927.
2. Ascroft, P. B.: The basis of treatment of vasospastic states of the extremities; and experimental analysis in monkeys. Brit. J. Surg., *24:* 787, 1937.
3. Brown, M. H.: Chemical sympathectomy in neurovascular lesions of the upper extremity. U. S. Nav. M. Bull., *46:* 343, 1946.
4. Fatherree, T. J., Adson, A. W., and Allen, E. V.: The vasoconstrictor action of epinephrine on the digital arterioles of man before and after sympathectomy. Surgery, *7:* 75, 1940.
5. Goetz, R. H., and Marr, J. A. S.: The importance of the second thoracic ganglion for the sympathetic supply of the upper extremities, with a description of two new approaches for its removal in cases of vascular disease. Clin. Proc., *3:* 102, 1944.
6. Goetz, R. H.: Collective review. Surgical physiology of sympathetic nervous system with special reference to cardiovascular disorders. Internat. Abstr. Surg., *87:* 417, 1948; in Surg. Gynec. & Obst. '48.
7. Grant, R. T.: Further observations on the vessels and nerves of the rabbit's ear, with special reference to the effects of denervation. Clin. Sc., *2:* 1, 1935.
8. Grimson, K. S., Wilson, H., and Phemister, D. B.: The early and remote effects of total and partial paravertebral sympathectomy on blood pressure. Ann. Surg., *106:* 801, 1937.
9. Grimson, K. S.: Sympathectomy and the circulation, anatomic and physiologic considerations and early and late limitations. Surgery, *19:* 277, 1946.
10. Hollinshead, W. H., and Finkelstein, H.: Regeneration of nerves to the adrenal glands. J. Comp. Neurol., *67:* 215, 1937.
11. Hyndman, O. R., and Wolkin, J.: Sympathectomy of the upper extremity. Arch. Surg., *45:* 145, 1942.
12. Hyndman, O. R., Wolkin, J., and Van Allen, M. W.: Cell stations in upper sympathetic chain. J. Neurosurg., *5:* 521, 1948.
13. Kirgis, H. D., and Ohler, E. A.: Regeneration of pre- and postganglionic fibers following sympathectomy of the upper extremity. Ann. Surg., *119:* 200, 1944.
14. Kuntz, A.: *The Autonomic Nervous System*, Ed. 3. Lea and Febiger, Philadelphia, 1945.
15. Lee, F. C.: The regeneration of nervous tissue. Physiol. Rev., *9:* 575, 1929.
16. Ray, B. S., Hinsey, J. C., and Geohegan, W. A.: Observations on the distribution of the sympathetic nerves to the pupil and upper extremity as demonstrated by stimulation of the anterior roots in man. Ann. Surg., *118:* 647, 1943.
17. Simmons, H. T., and Sheehan, D.: The causes of relapse following sympathectomy of the arm. Brit. J. Surg., *27:* 234, 1939.
18. Smithwick, R. H., White, J. C., and Freeman, N. E.: Effect of epinephrine on the sympathectomized human extremity. Arch. Surg., *29:* 759, 1934.
19. Smithwick, R. H.: Modified dorsal sympathectomy for vascular spasm (Raynaud's disease) of the upper extremity. Ann. Surg., *104:* 339, 1936.
20. Telford, E. D.: The technique of sympathectomy. Brit. J. Surg., *23:* 448, 1935.

TRAUMATIC LIVER INJURIES*

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The over-all mortality rate encountered with liver injuries has decreased during the past 40 years from 66.6 to 10 per cent.[18] This can be attributed to major advances in anesthetic techniques, blood transfusions, improved fluid and electrolyte therapy, antibiotics and our present mode of transporting the seriously injured patient. Earlier reviews consisted of reports of liver injuries due to blunt trauma, whereas more recent reviews deal with a much higher incidence of penetrating trauma.

This study was undertaken to evaluate the results of therapy for liver injuries encountered in a civilian hospital. During the period between January 1947 and January 1958, 189 patients with liver injuries were admitted to Grady Memorial Hospital, Atlanta, Georgia. This hospital serves as the primary emergency center for a population of approximately 800,000.

ANALYSIS

All but 2 of the 189 patients were treated surgically. The ages of the patients varied from 22 months to 71 years. Injuries were encountered predominantly in the 20- to 40-year age group with the mortality being higher in the younger (below 10 years) and older age groups (Table 1). There were 52 white patients and 137 Negro patients (ratio, 2.5:1).

No prolonged diagnostic measures were necessary in the majority of this group of patients. The mode of injury was usually indicative of abdominal visceral and often thoracic injury. Therapy was instituted usually immediately after the patients' arrival in the emergency clinics.

Intravenous fluid therapy, whole blood transfusions as indicated, nasogastric suction and, where indicated, closed thoracotomy instituted before surgery. In patients with stable vital signs, chest and abdominal films were obtained for aid in diagnosing pneumothoraces, free intraperitoneal air and localization of foreign bodies (bullets). Intravenous pyelography was utilized occasionally when renal damage was suspected.

In 167 patients the mode of injury was penetrating and in 22 the liver injury was due to a blunt or crushing injury (table 2). The liver wounds varied from a simple puncture wound to a massive maceration of liver substance as seen with the close range shotgun blast. Deep stellate fractures were the rule in patients sustaining blunt or crushing trauma.

Injury to associated organs was encountered in 120 patients. Pulmonary, gastric and renal injuries were more frequent and related primarily to penetrating gunshot wounds of the abdomen and chest (table 3). In many instances, injuries to other organs required more time and care during surgery than did the liver. The majority of the liver wounds had ceased bleeding at the time laparotomy was performed and required only inspection, evacuation of blood and blood clots and adequate drainage.

Whole blood replacement, in addition to parenteral fluids and plasma expanders, was required in 143 patients. Replacement ranged from 250 to 17,000 cc. As shown in table 4, the mortality rate can be correlated to the amount of whole blood administered to the patients. This is of course true in blood replacement for any cause.

The methods of treatment of the liver wounds were varied, depending upon the individual preferences of the surgical staff (table 5). Drainage was employed in only 126 patients explored. The use of gauze packs in an attempt to obtain hemostasis was unsuccessful. Diaphragmatic injuries were closed with interrupted sutures when encountered. Associated injuries were treated in an acceptable manner.

Postoperative management consisted of supportive measures, including a broad range of antibiotics. An over-all mortality of 16.4 per cent was encountered. A summary of the pertinent factors related to the fatalities is tabulated in tables 6 and 7. The mortality rate as shown here is not only related to the degree of liver trauma, but also to the number of associated organs

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TABLE 1

Mortality in age groups

Age	No. of Patients	Deaths	Per Cent
1–10	11	5	45.0
10–20	16	3	18.0
20–30	75	6	8.0
30–40	48	6	12.5
40–50	29	6	20.7
50–60	5	2	40.0
60–70	4	2	50.0
70–80	1	1	100.0
Totals	189	31	16.4

TABLE 2

Mortality related to mode of injury

Mode of Injury	No. of Patients	Deaths	Per Cent
Penetrating			
Ice pick	2	0	0
B.B. pellet	2	0	0
Knife	63	0	0
Broken glass	1	0	0
Shotgun*	15	3	20.0
Bullet	84	18	21.9
Blunt			
Auto accidents	17		
Direct blunt trauma	3	10	45.4
Falls	2		
Totals	189	31	16.4

* Five shotgun blasts were close ranged leading to massive maceration of liver substance.

TABLE 3

Associated organs injured

Organ	Mode of Injury				Total
	Stab	Gunshot wounds	Shotgun	Blunt	
Lung	12	27	5	9	53
Diaphragm	5	6	3	0	14
Stomach	1	25	3	1	30
Duodenum	1	14	1	1	17
Small bowel	2	11	1	2	16
Colon	2	15	3	0	20
Pancreas	2	12	0	3	17
Spleen	0	6	2	5	13
Gall bladder	1	9	0	0	10
Kidney	1	18	4	5	28
Major vessel	1	10	0	1	12
Heart	0	1	2	0	3
Brain	0	1	0	1	2
Spinal cord	0	5	0	0	5
Fractures (major)	0	2	2	8	12

TABLE 4

Mortality in relation to blood administration

Amount of Blood	No. of Patients	Deaths	Per Cent
cc.			
0	46	1*	2.7
500	40	3	6.5
1000	20	1	4.5
2000	37	3	8.1
3000	16	6	37.5
3000+	29	17	58.6
Totals	189	31	16.4

* Cardiac arrest with induction of anesthesia.

TABLE 5

Method of treatment for liver wounds

Type of Wound	No. of Patients	No. Repaired	Suture Only	Hemostatic Sponge Only	Suture and Hemostatic Sponge	Debridement	Resection	Drain
Perforating	86	45	13	14	12	2	0	68
Laceration	76	51	9	3	12	1	0	39
Stellate fracture	16	2	3	6	4	1	0	13
Macerating*	7	0	0	1	0	3	1	5
Puncture	4	4	0	0	0	0	0	1
Totals	189	102	25	24	28	7	1	126

* Gauze packs used in 2 macerating injuries.

TABLE 6

Summary of deaths resulting from liver injury

Case No.	Age	Mode of Injury	Nature of Liver Injury	Treatment of Liver Injury	Time of Death	Associated Injury	Remarks
1	50	Shot-gun	Multiple perforations	11,000 cc. blood, drainage	24 hr.	Spleen, lung, stomach, colon, pericardium	Massive blood loss
9*	18	Blunt	Laceration of dome	6500 cc. blood	15 days	Skull fracture, lungs, brain, mandible	Innominate vein erosion by tracheotomy tube
10	38	Blunt	4-cm. deep laceration	Gelfoam suture, 17,000 cc. blood	30 hr.	Spleen, kidney, ileum, lung, mesentery	Massive blood loss
13	62	GSW†	Perforation rt. lobe	Gelfoam suture, 3500 cc. blood	5 days	Both lungs, kidney, spinal cord	Lower nephron ne phrosis
23	31	GSW	Perforation, left lobe	1500 cc. blood, drainage	2 days	Pancreas, kidney	Hyperthermia to 107 cause unknown
26	4	Blunt	Stellate fracture, rt. lobe	500 cc. blood, drainage	3 hr.	Spleen, lung	Anoxia, massive pulmonary hemorrhage, cardiac arrest
30	22	GSW	Perforation, rt. lobe	4000 cc. blood, drainage	12 hr.	Duodenum, gall bladder, brain	Severe head injury
33	25	GSW	Perforation, rt. lobe	Gelfoam suture, 5000 cc. blood	3½ hr.	None	Massive hemorrhage, shock
36*	30	Blunt	7-cm. laceration, rt. lobe	500 cc. blood	8 days	Internal hernia	Bronchopneumonia, pericarditis
39	22 mo.	Blunt	Large laceration, rt. lobe	Gelfoam suture, 250 cc. blood, drainage	12 hr.	Chest (rib fracture)	Postop. circulatory collapse
45	16	Blunt	Multiple lacerations, rt. lobe	Gauze packs, 5000 cc. blood	1½ hr.	None	Massive blood loss
50	3	Blunt	Tear capsule, rt. lobe	350 cc. blood, drainage	24 hr.	Lung, spleen, kidney, radial fracture	Massive intrapulmonary hemorrhage
53	40	Blunt	Multiple stellate fracture	Gelfoam suture, 4000 cc. blood, drainage	4 days	Pancreas, renal vein	Lower nephron
64	44	GSW	Perforation, left lobe	5000 cc. blood, drainage	17 days	Pancreas, duodenum, vena cava	Multiple lung abscesses, peritonitis, abscess of head of pancreas
87	15	Blunt	8-cm. tear, left lobe; 5-cm. tear, rt. lobe	Gelfoam packs, 3500 cc. blood, drainage	25 days	Kidney, duodenum, pancreas, spleen	Peritonitis, biliary fistula (terminal bleeding, diathesis)
90	27	GSW	Superficial laceration, rt. lobe	4000 cc. blood, drainage	6 hr.	Lung, stomach, jejunum, pancreas, spleen	Massive trauma
91	56	GSW	Perforation, rt. lobe	Gelfoam, 3000 cc. blood, drainage	9 days	Duodenum, colon, vena cava	Pulmonary embolus
99	2	GSW	Perforation, left lobe	500 cc. blood, drainage	24 hr.	Stomach, small intestine, colon, lung, heart	Myocardial injury
122*	42	Blunt	4-cm. laceration, rt. lobe	3500 cc. blood	8 hr.	Fracture tibias, laceration of diaphragm, lungs, kidney, multiple rib fractures	Massive blood loss
124	31	GSW	Perforation, left lobe	Debridement, 4000 cc. blood, drainage	10 days	Stomach, duodenum, colon	Evisceration, lesser sac abscess
125	33	GSW	Perforation, left lobe	Debridement, Gelfoam, 2000 cc. blood	6 hr.	Lungs, spinal cord	Massive hemorrhage
128	48	GSW	Perforation, rt. lobe	Gelfoam, drainage	6 days	None	Anoxia during induction with cardiac arrest
131	26	GSW	Perforation, rt. lobe	Suture, 2000 cc. blood, drainage	10 days	Lung, kidney	Pulmonary embolus
147	42	GSW	Stellate fracture, left lobe	Gelfoam, 4500 cc. blood	12 hr.	Spleen, pancreas	Massive hemorrhage
155	23	GSW	Perforation, rt. lobe	3500 cc. blood	30 hr.	Jejunum, colon	Massive blood loss

TABLE 6 (*Continued*)

Case No.	Age	Mode of Injury	Nature of Liver Injury	Treatment of Liver Injury	Time of Death	Associated Injury	Remarks
161	21	GSW	Maceration, rt. lobe	Rt. lobectomy, 2500 cc. blood, gauze packs, drainage	4 hr.	Abdominal wall defect	Massive hemorrhage
166	48	GSW	Perforation, left lobe	2500 cc. blood, drainage	24 hr.	Lung, diaphragm, stomach	?Transfusion reaction
171	62	GSW	Perforation, rt. lobe	7500 cc. blood	24 hr.	Hepatic artery, spleen, kidney	Massive blood loss
173	71	GSW	Perforation, rt. lobe	3000 cc. blood, drainage	12 hr.	Colon, gall bladder	Massive hemorrhage
186	38	GSW	Rt. lobe massive destruction	Gelfoam, 2500 cc. blood, drainage	23 hr.	Duodenum, colon, kidney	?Transfusion reaction, massive blood loss
187	8	GSW	Multiple lacerations	Massive debridement and suture, 1000 cc. blood, drainage	20 hr.	Stomach, colon, jejunum, kidney, spleen	Massive trauma, pulmonary congestion, peritonitis, adrenal hemorrhage

• Liver injuries found at autopsy.
† GSW = gunshot wound.

injured and the blood loss resulting from the injuries.

The surviving 158 patients with liver injuries required an average of 36 days of hospitalization. One patient was hospitalized for a period of 97 days because of complications (pancreatic and duodenal fistulas) encountered; 83 major complications occurred in 65 patients surviving their extensive trauma (table 8).

Patients sustaining associated pancreatic, duodenal, or colon injuries were more prone to postoperative complications and a higher mortality rate (table 9). Only 4 patients developed biliary fistulas and these presented no prolonged or complicated postoperative difficulty.

DISCUSSION

The location, size and consistency of the liver contribute to its vulnerability to injury. The primary problem with these injuries is the re-

TABLE 7

Mortality related to number of organs injured

Organ Injured	No. of Patients	Deaths	Per Cent
Liver only	69	5	7.2
Liver and 1 organ	49	0	0
Liver and 2 organs	38	8	21.0
Liver and 3 organs	19	8	42.0
Liver and 4 organs	8	4	50.0
Liver and 5 organs	6	6	100.0
Totals	189	31	16.4

TABLE 8

Morbidity

Hemorrhage		1
Postoperative hemorrhage	1	
Infection		32
Subphrenic abscesses	11	
Wound abscess	12	
Drain tract (persistent)	2	
Pelvic abscess	1	
Empyema	3	
Thrombophlebitis	3	
Pulmonary		18
Atelectasis	11	
Late pneumothorax	1	
Recurrent hemothorax	5	
Bronchopleural fistula	1	
Gastrointestinal		5
Duodenal fistula	1	
Intestinal obstruction	2	
Prolonged ileus	1	
Jejunal fistula	1	
Biliary (fistula)		4
Pancreatic (fistula)		4
Genitourinary		5
Lower nephron nephrosis	4	
Ureteral stricture	1	
Wound (other than infection)		7
Dehiscence	5	
Evisceration	1	
Incisional hernia	1	
Miscellaneous		7
Pyelonephritis	2	
Serum hepatitis	1	
Acute parotitis	1	
Delirium tremens	2	
Fever, undetermined origin	1	
Total		83

TABLE 9

Morbidity and mortality related to associated pancreatic duodenal and colon injuries

Organ Injured	No. of Patients	Deaths	Fistula	Per Cent Complications
Pancreas	17	6	4	60
Duodenum	17	6	1	40
Colon	20	8	–	40

placement of blood loss and control of hemorrhage from the liver by surgical means. Beck[2] stated, in 1902, that "operations upon the organ were dreaded on account of the profuse, at times uncontrollable hemorrhage which followed the cutting into the very vascular organ." This still holds true to a great extent today. An increasing number of partial hepatic resections for hepatic lesions are now being reported in the literature.[15] Ponfick[14] and many other investigators proved by animal studies that resection of considerable portions of the liver is possible and compatible with life. Following massive trauma to the liver, the usual function tests of the liver are not greatly altered.[10]

Reduction in the mortality of liver wounds in the past 40 years has been accomplished by early diagnosis, adequate whole blood replacement, antibiotics, improved anesthesia and a recognition of the complications resulting from such injuries. With our present modes of transportation, the seriously injured patient can obtain much earlier resuscitative measures and definitive therapy at a medical facility.

Penetrating injuries of the thorax and abdomen are associated with a high incidence of liver involvement. Fox[5] reported 57 per cent liver involvement in a review of 270 thoracoabdominal wounds. Usually, liver wounds inflicted by stabbing with such weapons as knives and ice picks are not severe. However, injuries to associated organs by this means may be quite severe and lead to prolonged complications and an increase in the mortality rate. Duodenal, pancreatic and colon injuries markedly increase the incidence of complications encountered in liver wounds.

Gunshot wounds of the liver result in varying degrees of parenchymal damage. The explosive effect rather than the size of the missile is primarily related to the degree of injury. Close ranged shotgun blasts and crush injuries to the liver represent the most severe types of liver damage and are in turn associated with a much higher fatality rate.[17] This is demonstrated well by Mikesky and associates,[12] in their collective review of liver injuries with differentiation between penetrating and blunt trauma. Deep stellate fractures, partial to complete avulsion of large segments of liver tissue, and massive maceration are seen with blunt and shotgun blast injuries. Here, hemorrhage and its control is a major problem. Subsequent necrosis and sequestration of liver fragments may occur due to interference with the blood supply deep within the liver substance. Infection, biliary drainage and secondary hemorrhage are frequently encountered in this group of patients who survive initial surgical therapy.

The treatment in known or suspected liver injuries is early exploration. This is preceded by adequate resuscitative measures. The reconstitution or use of blood aspirated from the abdominal cavity has been reported and associated with a fatal result.[1] Adequate blood should be available during the course of the operative procedure.

The management of the liver wound will be dependent upon the magnitude of the injury. In over 50 per cent of the wounds encountered, active hemorrhage had ceased at the time of exploration. This has been cited by other authors.[18] Simple lacerations, unless actively bleeding, may be treated by drainage alone, provided the drainage is adequate. This is in disagreement with the views of Mikal and Papen[11] who advocate no drainage to sites of liver injuries because of possibilities of introduction of infection to the areas. If bleeding is active, suture of the laceration with blunt needles will provide adequate hemostasis. The use of autogenous material, such as muscle, omental fat and peritoneum, for aid in hemostasis has been advocated. Hemostatics such as gelatin sponge or foam are used more extensively with good results.[7] If these agents are utilized, they should be used sparingly.

Perforations of the liver with missiles may require nothing more than adequate drainage. The closure of both the entrance and exit wounds may prevent adequate drainage of the tract and thereby lead to intrahepatic accumulations of blood, bile, purulent material, or necrotic liver substance. Posttraumatic liver injuries have been associated with massive gastrointestinal bleeding (hemobilia) by several authors.[3, 6, 16, 19] No incidence of hemobilia was encountered in this

study. One is usually hesitant to embark on any extensive debridement of bullet tracts in the liver for fear of initiating a more extensive and uncontrollable hemorrhage.

Deep stellate lacerations or linear tears into the liver will require deep sutures to approximate the edges for hemostasis. Hemostatic agents within the sutured laceration may be necessary but should be avoided if possible. Absorption is prolonged and delays early healing of the laceration.[9] The placing of drains into the laceration may be advantageous in preventing intrahepatic accumulations of blood, bile and necrotic liver substance.[8] Partial or complete avulsion of large segments of liver substance usually require removal at the initial exploration; otherwise they may sequestrate and lead to undesired complications. Hemostasis can be effected with sutures through the remaining borders of the bare liver. Ligation of exposed large vessels should be performed. Hemostatic sponges may be incorporated over the bare area with sutures.

Extensive maceration or deep linear lacerations into the liver will require radical treatment. With control of the blood flow through the portal vein and hepatic artery by compression of these structures through the foramen of Winslow, partial resection or even lobectomy can be performed. Large gauze packs have been utilized but have not been successful because of the associated subsequent necrosis of liver tissue that develops, as well as an increase in intestinal obstruction.[4, 13]

Abdominal exploration should be complete with both penetrating and blunt or crush injuries to determine if associated organs have been injured. Pancreatic injuries alone are related to a high increase of complications. With free biliary leakage, pancreatic enzymes become activated and autodigestion of surrounding tissues ensues. Here again, after the injury has been repaired adequate drainage must be established. With liver injuries and associated organs involved, one must be cognizant of the increased incidence of complications that occur. With four or five organs injured the mortality rate rapidly reaches 80 to 100 per cent. This has been demonstrated not only in this study but in reviews previously reported.[12, 18]

SUMMARY

Traumatic liver injuries still constitute a major surgical problem. Morbidity and mortality are not only related to the degree of liver damage but also to the amount of blood loss, the number of associated organs injured, and specifically, associated injuries to the pancreas, duodenum and colon. The mortality rate in liver injuries resulting from blunt abdominal trauma is still high. The present methods of surgical treatment of liver injuries are adequate. Drainage from the site of injury is of prime importance. Until other methods of hemostasis are developed for liver injuries, radical debridement of the macerating or penetrating liver wounds will not be feasible.

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REFERENCES

1. Allen, A. W.: Internal injuries without penetrating wounds. New England J. Med., *205:* 34, 1931.
2. Beck, C.: Surgery of the liver. J. A. M. A., *38:* 1063, 1902.
3. Burnett, W. E., Rosemond, G. P., Caswell, H. T., and Hall, J. H.: Unusual case of gastrointestinal bleeding. Surgery, *26:* 1044, 1949.
4. Christopher, F.: Rupture of liver. Ann. Surg., *103:* 461, 1936.
5. Fox, D. B.: Thoracoabdominal wounds. Surg. Gynec. & Obst., *82:* 64, 1946.
6. Hawthorne, H. R., Oaks, W. W., and Neese, P. H.: Liver injuries with a case report of repeated hemorrhage through biliary ducts. Surgery, *9:* 358, 1941.
7. Jenkins, H. P., Janda, R., and Clarke, J.: Clinical and experimental observations on the use of gelatin sponge or foam. Surgery, *20:* 124, 1946.
8. Krieg, E. G.: Hepatic trauma. Arch. Surg., *32:* 907, 1936.
9. Madding, G. F.: Injuries of the liver. A. M. A. Arch. Surg., *70:* 748, 1955.
10. Martin, J. D., Jr.: Wounds of the liver. Ann. Surg., *125:* 756, 1947.
11. Mikal, S., and Papen, G. W.: Morbidity and mortality in ruptured liver. Surgery, *27:* 520, 1950.
12. Mikesky, W. E., Howard, J. M., and DeBakey, M. E.: Injuries of the liver in 300 consecutive patients. Internat. Abstr. Surg., *103:* 323, 1956.
13. Pilcher, L. S.: Massive rupture of the liver. Ann. Surg., *116:* 827, 1942.
14. Ponfick, V. A.: Cited by Beck.[2]
15. Quattlebaum, J. K.: Massive resection of the liver. Ann. Surg., *137:* 787, 1953.
16. Sandblom, P.: Hemorrhage into the biliary tract following trauma—"traumatic hemobilia." Surgery, *24:* 571, 1948.
17. Sanders, G. B., Macguire, C. H., and Moore, R. H., Jr.: Massive rupture of the liver. Am. J. Surg., *78:* 699, 1949.
18. Sparkman, R. S., and Fogelman, M. J.: Wounds of the liver. Ann. Surg., *139:* 690, 1954.
19. Sparkman, R. S.: Massive hemobilia following traumatic rupture of liver. Ann. Surg., *138:* 899, 1953.

THE MANAGEMENT OF ENDOMETRIOSIS OF THE COLON*

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The general surgeon will not be called upon to give the primary diagnosis and treatment of endometriosis as often as the gynecologist. However, the frequent finding of endometriosis at laparotomy, suspected preoperatively or not, requires that anyone doing abdominal surgery be alert to the possibility and implications of endometriosis. This situation will present itself most often when the pathologic process involves the adjacent bowel. Accordingly, this paper will deal only with endometriosis involving the colon.

This disease process has been familiar to the gyneclologists for more than 50 years. The earliest accurate descriptions were by Von Recklinghausen and Cullen before 1900. They both described the "chocolate cysts" and felt that they were not malignant but were related to normal endometrial tissue. It was believed that these tumors developed from embryonic "rests." This theory was unchallenged until 1921, when Sampson proposed his theory of retrograde menstruation, which, in spite of its many protagonists and antagonists, has been the most widely accepted explanation. Experimental procedures have been performed which certainly substantiate such a theory. However, distant extraperitoneal sites of the disease give support to those who champion others of the numerous theories of development.

INCIDENCE

The incidence of proved endometriosis varies greatly in the current literature, seemingly dependent on many factors. In consecutive laparotomies the percentage of those with manifest endometriosis is generally reported at about 8 to 10 per cent.[8] However, in consecutive laparotomies performed on a private gynecology service, this incidence rises from 13 per cent[5] to 35 per cent.[14] There is universal agreement, without adequate explanation, that the incidence of endometriosis in ward patients is much less than in private patients. Meigs[14] reported an incidence of 8.1 per cent in ward patients and Johnson and associates[11] an incidence of 6.5 per cent in a similar group. Endometriosis in the Negro is rare indeed, and it is postulated by Boles and Hodes[2] that a high incidence of salpingitis in this group is responsible.

In those patients who were found to have external endometriosis, the large bowel, primarily sigmoid colon and rectovaginal septum, were involved in from 34 per cent[12] to 40 per cent.[10] In this latter report there was some degree of obstruction present in 45 per cent of those patients with involvement of the rectosigmoid. The incidence of complete obstruction secondary to endometriosis is quite low but this does occur.

PATHOLOGY

The lesions of external endometriosis are in reality islands of endometrial tissue with functioning glands and stroma under the direct control of the ovarian hormones. This includes regular episodes of menstruation, the irritative products of which cause a marked inflammatory response and localized fibrosis. The disease is characteristically found on the serosal surfaces of the pelvic viscera but the list of general abdominal organs with primary involvement is lengthy. In addition, distant sites of endometriosis have been reported, including kidney, lung, muscle and skin.[3, 9]

This disease may manifest itself either directly or indirectly. In the former instance, the so-called "endometrioma" directly involves the specific organ and by its periodic engorgement, with resultant pressure, may cause cyclic pain or obstruction of a hollow viscus, *i.e.*, bowel, or ureter. There may also be direct manifestations caused by the rupture of a surface lesion with irritation of the peritoneum by the products of menstruation. Indirectly these surface lesions may involve any nearby structure, becoming firmly adherent. This process may result in partial or complete obstruction of adjacent bowel and as the disease progresses this bowel may become directly involved by the growing endo-

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metrial tissue. This growth usually stops short of the lining mucosa and bleeding into the lumen from frank ulceration is uncommon, if not rare.[1]

By those most familiar with endometriosis. the disease is felt to meet the microscopic criteria for benignancy, but to develop and spread by extension and implantation as well as by lymphatic and venous embolization, just as cancer.[15]

DIAGNOSIS

The diagnosis of endometriosis of the large bowel is made clinically in a patient only if the surgeon is alert to the general symptomatology of external endometriosis in the patient with an unknown lesion of the bowel. This symptom complex includes dysmenorrhea (especially when progressive), dyspareunia and low backache, all of which are exaggerated in the postovulatory phase of the menstrual cycle. The patient usually has abnormal uterine bleeding and is sterile. In addition, she frequently has tender nodules in the uterosacral ligaments, fixed retrodisplacement of the uterus, and cystic ovaries.[5]

In those cases with bowel symptoms, certainly sigmoidoscopy and barium enema are indicated. Sigmoidoscopy may be of decreased value because of the frequency of severe pain with any manipulation and the low incidence of mucosal involvement.[3, 13] The barium enema is felt to be of considerable value to all and felt to be diagnostic by some[17] (table 1).

The clinical decision that the specific bowel lesion is due to endometriosis or cancer requires exploration for confirmation. However, if the general symptomatology of endometriosis is present with barium enema confirmation and few of the symptoms of cancer, this may be a reasonable presumptive diagnosis.

TABLE 1

Chief diagnostic features of endometriosis on barium enema[17]

1. Intact mucous membrane
2. Fixation of the bowel
3. Tenderness on fluoroscopy near menstrual period
4. Colon otherwise normal
5. Lesion may be (*a*) concentric, or stenosing (*b*) polypoid
6. Usually sharp demarcation with concentric lesions
7. Obvious involvement of bowel wall with polypoid defect
8. Usually short length
9. Exacerbation of findings at menstrual period

FIG. 1 Photograph at operation showing left ovary adherent to sigmoid colon.

CASE REPORT

N. J. (S.J.I. 61125), a 38-year-old housewife, was admitted to the hospital because of abdominal pain. She gave a history 15 years in duration of similar, though less severe, left lower quadrant pain associated with episodes of diarrhea and alternating with longer periods of constipation. These symptoms were ascribed to "colitis," and were frequently associated with her menses. Review of systems was negative except for sterility and a history of a "tilted" uterus, which had not responded to conservative measures. Physical examination revealed moderate lower abdominal tenderness with a tender uterus fixed in retroflexion.

Her pain subsided and barium enema revealed a smooth filling defect at the junction of the descending and sigmoid colon. Sigmoidoscopy was negative to 20 cm. A tentative preoperative diagnosis of endometriosis or sigmoid neoplasm was made and treatment discussed with the patient, who refused castration. At laparotomy, external endometriosis with endometriosis of the sigmoid was found. A local resection of the colon lesion was performed. The patient had an uneventful convalescence and has been well since (fig. 1).

MANAGEMENT

The management of external endometriosis without bowel involvement ranges from "watchful waiting" for the clear-cut and relatively mild cases to total hysterectomy with bilateral sal-

pingo-oophorectomy for advanced cases with no desire to preserve childbearing ability. Conservative surgery, which first establishes the diagnosis and secondly removes the smallest portion of reproductive tissue possible to control the disease, is the most satisfactory initial procedure.[5, 7] The possibility that this approach may prove inadequate and reoperation for complete removal of the reproductive organs may be necessary, is felt justified if the patient who is anxious for children has had this possibility explained before the first procedure. Radiation castration is used only in those cases of recurrence after the diagnosis has been established at earlier laparotomy. Hormone therapy has but few vigorous advocates, and many critics.[4]

The plan for management of endometriosis involving the colon will depend on two factors: (1) the extent of the lesion, whether it is simply a serosal implant or is one with clinical or radiological signs of partial or complete obstruction; and (2) the decision as to the type of treatment for the general endometriosis will influence the treatment of the bowel lesion. For example, where conservation of ovarian function is elected, superficial bowel lesions may be excised completely without difficulty. If, however, there is more than superficial involvement of the bowel in this situation, resection of the involved bowel should be performed for microscopic confirmation of the diagnosis as well as for relief of the obstructive symptoms. This was the procedure elected in the case report.

Conversely, where ablation of ovarian tissue is elected, superficial bowel lesions may be left in place or excised for frozen section if their exact nature is uncertain. Where the colon lesion is more extensive there are two groups of opinion. The first group feels that resection is not necessary if the diagnosis is ascertained by frozen section.[7, 9, 10] Even with partial or complete obstruction the symptoms may be relieved within a period of days by castration alone.[1] The second group feels that by not removing the lesion the presence of a carcinoma may be overlooked, in spite of a negative frozen section. They also feel that more ultimate distortion and scarring will occur than with resection of the lesion.[6, 11, 13, 16]

The author feels that the latter approach is the more reasonable, for the reasons mentioned, together with the paucity of complications resulting from the local resection of colon well prepared preoperatively.

SUMMARY

1. A review of the history, pathology, incidence and diagnosis of endometriosis of the colon is presented, with an illustrative case.

2. The management of such lesions is discussed, with particular reference to the place of bowel resection.

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REFERENCES

1. Benz, E. J., Dockerty, M. B., and Dixon, C. F.: Polypoid endometrioma of the colon. Proc. Staff Meet. Mayo Clin., *27:* 201, 1952.
2. Boles, R. S., and Hodes, P. J.: Endometriosis of the small and large intestine. Gastroenterology, *34:* 367, 1958.
3. Charles, D.: Endometriosis and hemorrhagic pleural effusion. Obst. & Gynec., *10:* 309, 1957.
4. Cooke, W. R.: Observations on the massive stilbesterol therapy of endometriosis. Am. J. Obst. & Gynec., *71:* 569, 1956.
5. Depp, O. R., and Pope, D.: Early conservative surgery in endometriosis. South. M. J., *49:* 1345, 1956.
6. Ferraro, L. R., Hetz, H., and Carter, H.: Malignant endometriosis. Obst. & Gynec., *7:* 32, 1956.
7. Gardner, G. H.: Management of pelvic endometriosis. Obst. & Gynec., *5:* 538, 1955.
8. Green-Armytage, V. B.: Endometriomata: the five clinical types. Brit. M. J., *1:* 602, 1933.
9. Henriksen, E.: Endometriosis. Am. J. Surg., *90:* 331, 1955.
10. Jenkinson, E. L., and Brown, W. H.: Endometriosis. J. A. M. A., *122:* 349, 1943.
11. Johnson, C. G., Coppola, A. F., and Moll, C. F.: Complications of endometriosis of the sigmoid colon. South. M. J., *50:* 893, 1957.
12. Kratzer, G. L., and Salvati, E. P.: Collective review of endometriosis of the colon. Am. J. Surg., *90:* 866, 1955.
13. McMahon, J. M., and Shutkin, M. W.: Clinical pathological conference from the Tours Infirmary, New Orleans. Am. J. Gastroenterology, *29:* 657, 1958.
14. Meigs, J. V.: Endometriosis. Ann. Surg., *127:* 795, 1948.
15. Melody, G. F.: Endometriosis: a half-century of progress. Stanford M. Bull., *13:* 125, 1955.
16. Melody, G. F.: Endometriosis causing obstruction of the ileum. Obst. & Gynec., *8:* 468, 1956.
17. Wietersen, F. K., and Balow, R. M.: Colonic endometriosis: roentgenologic aspects. Radiology, *69:* 839, 1957.

INJURIES OF THE KIDNEY*

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The temperament of the present age, the eager participation in sports, and the increasing number of mechanical contrivances of speed have combined to cause mankind a greater incidence of bodily trauma. Accordingly, a review of the experiences in 137 cases of injury to the kidney admitted to Grady Memorial Hospital, Atlanta, Georgia, during the 10-year period between December 1947 and January 1958 seemed timely. For the most part these patients were diagnosed and treated by the house staff under advice and supervision of the attending surgeons.

Most injuries to the kidney occur during the 3rd and 4th decades. In this series there were 6 under 10 years, 26 above the age of 40, and 105 between ages 11 and 39 (table 1).

Males predominated over females in the ratio of 3½ to 1. Due to the large segment of Negro population with their potential for physical encounter in this area, the colored race predominated, there being 98 colored to 39 whites, a proportion of 2½ to 1 (table 2).

The left side was involved slightly more than the right (table 3). In the series reported by Crane and Knigge,[2] a similar slight dominance of the left side existed.

Various types of trauma are responsible for injuries to the kidney. This particular series assumes a unique pattern, since 53 per cent of the injuries were attributed to penetrating wounds; the majority were caused by gunshot and pistol bullets with a small percentage contributed by stabbings (table 4). In a report of 220 kidney injuries,[2] only 10 per cent were caused by penetrating wounds. Automobile accidents accounted for 25 per cent; the remaining were due to falls, industrial mishaps and accidents coincident to sports.

Due to the large number of gunshot wounds in which the multiple injuries to the intraabdominal viscera necessitated emergency exploration, 53 (38 per cent) were not subjected to the customary diagnostic procedures. The extent of the injury to the kidney was recognized and treated at laparotomy. The diagnosis in the remainder was estimated by cystoscopy, excretory urography, retrograde studies or various combinations of these procedures (table 5).

The degree of injury was classified as contusions, minor and major lacerations with and without extravasation of urine. A contusion implied no loss in continuity of the renal parenchyma or the true capsule of the kidney. There was some intrarenal hemorrhage of a mild degree rarely associated with a minor subcapsular tear. Gross or microscopic hematuria was present. Minor lacerations were arbitrarily classified as having small breaks in the parenchyma and the capsule with the accumulation of 100 cc. or less of blood adjacent to the kidney, as judged by x-ray or as observed at exploration. Major lacerations gave evidence of bleeding in amounts greater than 100 cc., presented a palpable tumor on physical examination, and provided evidence by x-ray of obliteration of the anatomical landmarks in the area of the afflicted kidney. Extravasation, as the word implies, was characterized by leakage of urine around the kidney. In this situation contrast media was demonstrated by pyelography to extend beyond the confines of the calyceal system. In some instances extravasation could not actually be demonstrated when a torn calyx plugged by a firm clot prevented leakage at the time the pyelogram was made.

The majority (47 or 34 per cent) were classed as contusions, but minor and major lacerations with and without extravasation totaled 87 (62 per cent). These were: 29 (21 per cent) minor lacerations, 27 (19 per cent) major, and 31 instances of extravasation (22 per cent). Direct trauma to the renal pedicle occurred in 8 patients and, except for 2 cases, was associated with laceration of the parenchyma (table 6). Seven patients were noted to have had pre-existing disease of the injured kidney.

Penetrating wounds of the kidney are frequently accompanied by serious injury to the intraperitoneal structures. Frequently there is involvement of the spleen, liver, bowel and

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TABLE 1

Age (Years)	Number of Cases	Per Cent
Less than 10	6	4
11–20	27	20
21–30	48	35
31–40	30	22
41–50	14	10
51–60	4	3
61–70	7	5
71–80	1	1

TABLE 2

Sex	Race	Number of Cases	Per Cent
Male	White	32	23
Male	Colored	75	55
Female	White	7	5
Female	Colored	23	17

TABLE 3

Side	Number of Cases	Per Cent
Right	55	40
Left	74	54
Bilateral	3	2
Not recorded	5	4

TABLE 4

Mode of Injury	Number of Cases	Per Cent
Penetrating wounds	73	53
Crushing (auto, train)	35	25
Falls		
Buildings	6	
Miscellaneous	6	9
Direct Contact		
Football	3	
Fights	3	12
Direct blows	10	
Unrecorded	1	1

pancreas. The bleeding from these structures is profuse and prolonged, commanding priority in surgical aid. The contiguous ribs and transverse processes are commonly fractured (table 7).

In this series, 64 were treated medically whereas the remaining 73 were subjected to surgery.

TABLE 5

Method of Diagnosis	Number of Cases	Per Cent
Intravenous pyelography	49	36
Retrograde pyelography	8	6
Combined intravenous and retrograde	19	14
Abdominal exploration alone	53	38
Clinical impression	5	3
Cystoscopy, observation of bladder and clinical impression	2	2
Autopsy	1	1

TABLE 6

Pathology	Number of Cases	Per Cent
Contusions	47	34
Lacerations, minor	29	21
Lacerations, major	27	19
Lacerations with extravasation	31	22
Involvement of renal pedicle	8	5
Unknown	1	1

TABLE 7

Associated Injuries	Number of Cases	Per Cent
Fractures		
Skull	5	4
Ribs	20	15
Extremities	17	12
Pelvis	6	4
Transverse processes	12	9
Visceral		
Liver	31	23
Spleen	10	7
Pancreas	13	9
Bowel or mesentery	35	25
Diaphragm	11	8
Lung and pleura	11	8
Bladder	1	1
Ureter	1	1
Aorta, vena cava, portal vein	3	2
No associated injuries	46	34

Of the 73 operations, there were 60 laparotomies for exploration of coincident intraperitoneal trauma. In 13 operations the injury was confined to the kidney and exploration performed

TABLE 8

Treatment	Number of Cases	Per Cent
Renal injury *per se* without involvement of other viscera		
Medical	64	46
Repair laceration, drainage	1	1
Nephrectomy alone	12	9
Renal surgery coincident to laparotomy for suspected visceral pathology		
Nephrectomy	20	14
Partial nephrectomy	1	1
Repair laceration plus drainage	12	9
Drainage alone	23	17
Injury recognized, nothing done	3	2
Unknown procedure	1	1

for lacerations with or without extravasation. Of these 13 surgical procedures, the injuries proved to be so severe that nephrectomy was required in 12. Expectant observation was employed in 16 cases of laceration (table 8).

Studies of the survival rate of the 137 cases disclosed that 110 lived and 27 died. Of the 27 who died, there were 19 who succumbed in less than 24 hours following admission to the hospital. Of the 27 deaths, 20 were attributed to associated injuries or their complications. In 4 instances the main factor in the mortality was the injury to the kidney, but associated extrarenal structures were involved. In 3 cases direct relationship between the injury to the kidney and the mortality was established (table 9).

DISCUSSION

The diagnosis of renal injury is relatively easy. The history of trauma, pain in the side, possible palpable tumor, muscle guarding, and the presence of blood in the urine alerts one to the condition. What is of greater importance is to determine the extent of the injury, the degree of laceration, and whether or not extravasation of blood and urine is present. The specific treatment, in each instance, hinges upon the summation of these factors.

Those patients with evidence of intraperitoneal injury need not be subjected to extensive urologic diagnosis. Exploration would reveal the magnitude of the renal pathology. However, if circumstances permit, the presence and adequate function of the uninjured kidney as determined by excretory urography would be comforting knowledge. When the injuries seem confined to the kidney and surrounding skeletal structures (ribs, transverse processes, musculature of the abdominal wall), more deliberate attempts to appraise the extent of the injury should be undertaken. A difference of opinion exists as to whether or not to perform retrograde pyelography or to base the decision regarding treatment solely on the interpretation of the excretory urograms. Prather[4] reported that in 2 of 8 cases excretory urograms were misleading. Sargent and Marquardt[6] favor ascending pyelography whereas Priestley and Pilcher[5] state that cystoscopy and pyelography are often unnecessary and they are inclined to place much reliance on intravenous urograms. After treatment for shock has restored the blood pressure to systolic level of 90 or higher, excretory urography may offer some information on the traumatized side. However, the knowledge gained may be equivocal or insufficient; the contrast media may not be excreted, or be much delayed. At times the uninjured half or two-thirds of the kidney may excrete, appear normal, and yet one of the poles could be completely or partially separated. It is mandatory to arrive at

TABLE 9

Mortality	Number of Cases	Per Cent
Lived	110	80
Died	27	20
Died less than 24 hours after admission	19	14
Death due to associated injuries or complications	20	15
Death mainly from renal injury but with associated injuries	4	3
Death directly attributed to renal injury	2	2
*Renal injury *per se* with surgery	2	2
†Renal injury *per se* without surgery	1	1

* In one case the patient died on eighth postnephrectomy day, from lower nephron nephrosis; in the second case, abdominal exploration for stab wound with massive renal bleeding, the patient died from shock after 6 hours.

† Died on eighth day from secondary renal hemorrhage.

FIG. 1. Excretory urogram illustrating intact left kidney, good function on right. Extravasation not demonstrated.

a specific decision as to whether or not to perform emergency surgery or to elect expectant observation. The employment of cystoscopy and ascending pyelography furnish the details required for this decision. The presence or absence of extravasation would be determined, the degree of retroperitoneal hemorrhage characterized by the presence of a mass or displacement of the kidney by means of the x-ray could be estimated. The possibility of initiating bleeding from the pressure effects of retrograde injection on the side of the injury is slight if it exists at all. The inciting of infection by the introduction of the catheter to the area of trauma is worthy of consideration, but clinical experience indicates a minimal risk. Certainly, the information gained far outweighs the dangers to the patient. The information gathered by combined excretory and retrograde studies overshadows the impression of the clinical condition of the patient. If judgement pertaining to the decision to elect surgery were based solely on the clinical status plus excretory urography a grave error may be committed. The following case report serves to illustrate the importance of ascertaining complete appraisal of the extent of injury (figs. 1–4).

CASE REPORTS

Case 1. In an attempt to break up a fight, a 25-year-old colored woman sustained a blow to the right side of the back. Her next voiding was grossly bloody, but the urine appeared normal by the following day. She attended her usual duties and was asymptomatic except for vague discomfort in the loin. A gradual increase in the degree of pain provoked a visit to the hospital 5 days following the injury. She was uncomplaining, comfortable, had moderate degree of pain in the back, but no palpable tumor. Her pulse, blood pressure and temperature were normal. The excretory urogram (fig. 1) revealed an intact left upper urinary tract. On the right side the upper calyceal system was clear cut and normal, the middle and lower group not visualized. No extravasation seemed evident. Subsequent retrograde pyelography (fig. 2) disclosed extravasation of contrast media with presence of a mass in this area. On the basis of the findings of retrograde pyelography, surgical ex-

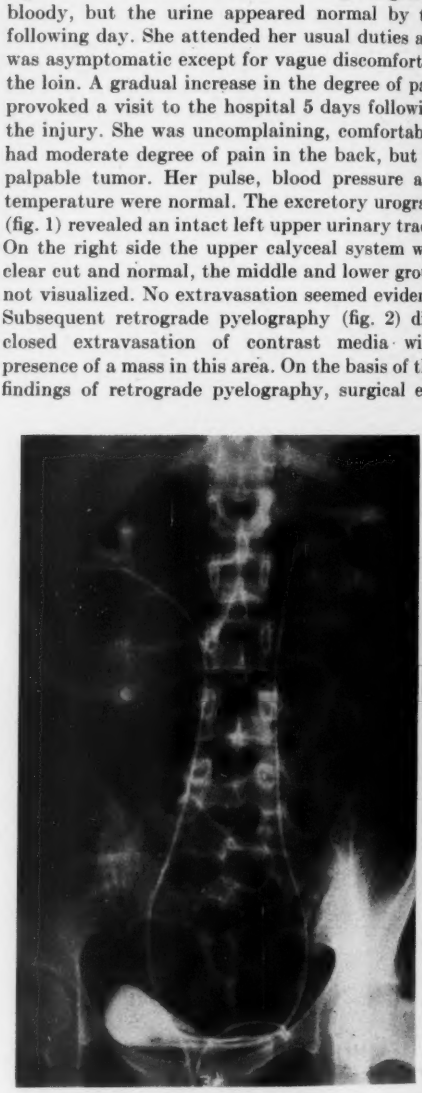

FIG. 2. Retrograde pyelogram showing contrast medium extravasating outside the collecting system.

ploration with right nephrectomy was performed with recovery. The removed kidney showed a large laceration extending into the pelvis and hilum almost dividing the organ in half (figs. 3 and 4).

Comment. The tendency toward treatment by conservative medical observation is readily comprehended and, in cases classed as contusions, is the method of choice. The intact capsule, minimal renal disruption, intimates healing will occur. The clinical experience gained from this series of 137 cases suggests exploration is indicated in all instances of injury to the kidney other than contusions. When there is extravasation of urine or massive hematoma, operation should be scheduled. Indecision may arise where laceration, major or minor, without demonstrable extravasation exists. There were 56 (40 per cent) such situations in this series. After stabilization of the patient's circulatory system which ordinarily requires 24 to 48 hours, and if coexisting injuries do not contraindicate, renal surgical exploration should be undertaken. The exposure would permit the surgeon to fully appraise the situation and apply definitive measures. Hemorrhage could be arrested, hematomas evacuated, unrecognized extravasation would be afforded drainage. Infarcted areas could be cleanly resected, lacerations sutured, and delayed infection minimized by access to drainage. At times a kidney might be salvaged that otherwise may demand delayed nephrectomy. Fullerton[3] found 22 per cent of the cases of renal injury had life jeopardized by the complication of delayed secondary renal hemorrhage occurring anytime up to 3 weeks. Immediate nephrectomy was the only treatment of choice and served as a life-saving measure. Furthermore, the late sequelae related to inadequately treated injuries may well be avoided. Colston and Baker[1] thoroughly studied 13 patients who had previously sustained injury to the kidney and each of which had been

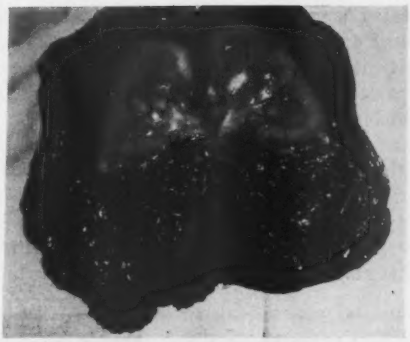

FIG. 4. Kidney bisected illustrating extensiveness of rupture and clot encasing the entire kidney. Note intact upper half of parenchyma.

FIG. 5. Retrograde pyelogram on right, calcified mass on left. Catheter would not pass beyond this level.

FIG. 3. Kidney removed by nephrectomy enveloped by blood clot except upper medial portion.

treated conservatively. His investigations disclosed 4 with calcified perirenal cysts, 2 locular cysts attached to the kidney, 1 hydronephrosis, 1 stone with hydronephrosis, 2 with calculus, 1 with hydrocalyx, 1 of delayed extravasation of urine and 1 with chronic infection associated with calyceal distortion. In each case in his series, a definite relationship was established between the trauma and the late complications described. The following case report illustrates what may ultimately occur to an injured kidney.

Case 2. This 52-year-old white woman injured her back at age 10. She recalled having been quite ill but did not recollect any specific diagnosis. She probably suffered a laceration of the kidney which was not recognized at the time. The calcified mass seen on the x-ray (fig. 5) was removed 43 years following the injury. The ureter was imperforate where it was attached to the mass and the latter was a cystic sac with a calcified wall which contained necrotic epithelial debris and crystals of fat without any elements of residual kidney.

Comments. Grady Memorial Hospital is a metropolitan institution with an extremely active accident service. Many of the patients are irresponsible and unintelligent, precluding the possibility of careful follow-up studies for the detection of delayed sequelae. However, 4 out of 40 who returned for various other complaints were found to have developed hypertension of a severe degree. Each of these patients was normotensive at the time of the original injury. Each injury was treated "conservatively." Of the 4, 1 who had sustained a stab wound of the kidney 3 years previously succumbed to hypertension. She possessed a small contracted kidney on the injured side. The remaining 3 could not be urged to return for further study.

CONCLUSION

In conclusion, may it be suggested that when possible, complete diagnostic studies in every case of injury to the kidney should be pursued. Surgical exploration after adequate measures have succeeded in reversing shock is generally urged in all cases other than contusions.

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REFERENCES

1. Colston, J. A. C., and Baker, W. W.: Late effects of various types of trauma to the kidney. Tr. Am. A. Genito-Urin. Surgeons, *28:* 171, 1935.
2. Crane, J. F., and Knigge, W.: Table, quoted from Campbell, M. F., *Text of Urology*, Vol. 2, p. 865. W. B. Saunders Company, Philadelphia, 1954.
3. Fullerton, A.: Quoted by Gordon-Taylor, G.: Complicated injuries of the urinary tract. Brit. J. Urol., *12:* 1, 1940.
4. Prather, G. C.: Traumatic conditions of the kidney. J. A. M. A., *114:* 207, 1940.
5. Priestley, J. T., and Pilcher, F., Jr.: Traumatic lesions of the kidney. Am. J. Surg., *40:* 357, 1938.
6. Sargent, J. C., and Marquardt, C. R.: Renal injuries. J. Urol., *63:* 1, 1950.

LEIOMYOSARCOMA OF THE DUODENUM: A SOURCE OF MASSIVE INTESTINAL HEMORRHAGE; REPORT OF TWO CASES*

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Gastrointestinal hemorrhage from leiomyosarcoma responds poorly to conservative therapy. The tumor grows slowly and can be treated surgically, often with only simple excision of the lesion. Although admittedly rare, they do not occur as infrequently as heretofore supposed. The major impediment to adequate therapy seems to be failure to recognize the lesion. This is based on the lack of initial symptoms, difficulty in demonstrating an extraluminal tumor by x-ray studies, and mimicking of peptic ulcer symptomatology.

A fatality resulted in a patient with a readily resectable lesion who presented with massive gastrointestinal hemorrhage. Another patient with intestinal hemorrhage responded to palliative resection of a similar lesion. With suspicion of a duodenal tumor as a possible cause of massive gastrointestinal bleeding, further fatalities from this lesion may be prevented. For this reason and because of the paucity of similar reports in the literature, these two cases and a review of the literature are presented.

CASE REPORTS

Case 1. A 65-year-old Negro man was admitted to the medical service of the Grady Memorial Hospital on January 18, 1956, with a chief complaint of weakness and epigastric pain. The patient was seen on a routine visit to the Cardiovascular Clinic on January 18, 1956, with similar complaints. Bouts of nausea and vomiting had occurred during the past 3 weeks. He denied hematemesis but stated that he had had several grossly bloody stools 3 days before this admission. The hemoglobin determination was 3.3 gm. per cent.

The patient had been seen in the outpatient clinic since 1949 for marked hypertension. In 1954, he had an episode similar to his present illness with a hemoglobin drop to 7.3 gm. per cent. He was placed on an ulcer regimen of antispasmodics and alkali which relieved his pain. A gastrointestinal series was negative. His hemoglobin returned to a level of 13.0 gm. per cent on iron therapy.

On admission to the hospital the pulse rate was 100; respiration, 24; and blood pressure, 140/82. Grade III eyeground changes, mild venous distension of the neck veins in a supine position, and cardiomegaly were noted. Moist rales were heard in both lung bases. The abdomen was obese with mild epigastric tenderness, normal peristalsis, and no palpable organs or masses. Rectal examination disclosed feces of a dark red, current jelly appearance. The remainder of the examination was negative.

Therapy instituted on admission consisted of an ulcer diet, sedation and digitalis. Because of his congestive failure and previous history of cardiac difficulties, he was given 900 cc. of packed red cells. His hemoglobin rose to a level of 10.0 gm. per cent. On the second hospital day he had two grossly bloody stools with a drop in hemoglobin to 7.2 gm. per cent. Another 300 cc. of packed red cells were given with a hemoglobin rise to 9.1 gm. per cent. His vital signs remained unchanged from those recorded on admission. In the afternoon he complained of severe epigastric pain followed by a drop in hemoglobin to 6.3 gm. per cent. He was then given 1000 cc. of whole cross matched blood, without evidence of developing pulmonary edema.

On the third hospital day he received another 300 cc. of packed red cells and 500 cc. of whole blood. He was then transferred to the surgical service and an emergency gastrointestinal series was obtained. This examination revealed no abnormality. He was returned to the ward with a blood pressure of 130/80 and a pulse of 88 per minute. On the basis of his cardiac difficulties, continued conservative measures were favored.

The patient rested quietly with stable vital signs until 3:00 a.m. of the morning of the fourth hospital day, when he suddenly had a large black liquid stool of approximately 400 cc. Administration of another 500 cc. of whole blood was started in the lower saphenous vein. His pulse rose to 110 and became irregular. His blood pressure dropped to 110/70. At 4:15 a.m. he had another liquid stool of approximately 700 cc. of

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FIG. 1. Leiomyosarcoma of the second portion of the duodenum with mucosal ulceration. This lesion was found at postmortem examination in case 1. Note the extralumenal mass. The probe is in the ulceration from which the fatal hemorrhage occurred.

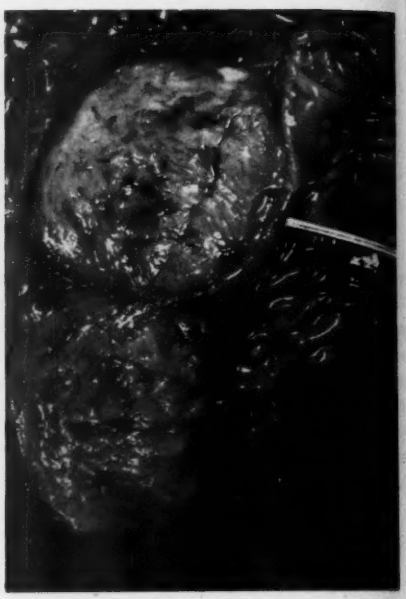

FIG. 2. This is the same lesion as shown in figure 1, with the tumor bisected through the ulceration. Note the small sinus tracts that open into the ulcer bed.

bright red blood. Following this he became cold, pale and clammy, with his blood pressure becoming imperceptible. He did not respond to blood given under pressure through a radial vein cut-down and was pronounced dead at 5:40 a.m. His hemoglobin concentration, determined shortly before his death, was 8.5 gm. per cent and the hematocrit was 28 volume per cent.

Postmortem examination revealed a 4 by 3 by 3-cm. tumor on the lateral border of the second portion of the duodenum 10 cm. from the pylorus. The tumor presented subserosally but extended through the muscle layers to form a 1 by 3-cm. ulceration on the mucosal surface (fig. 1). The tumor was rubbery in consistency and upon sectioning had a fibrous appearance with a central necrosis and sinus formation (fig. 2). The tumor was fairly well encapsulated. No metastatic lesions were found.

Histologic examination revealed irregular whorls of spindle shaped cells with moderate anaplastic features of the nuclei. The nuclei had blunt ends and in some places exhibited palisading. The tumor extended into the submucosa and contained many scattered mitoses (figs. 3 and 4).

FIG. 3. This low power view of the sarcoma demonstrates invasion of the submucosa.

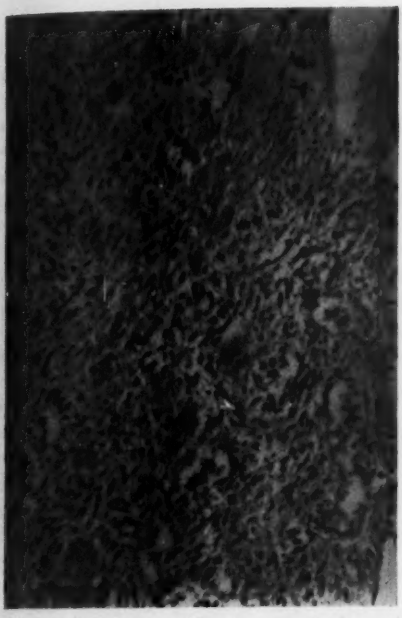

FIG. 4. Greater magnification reveals the variation in the cellular pattern of the sarcoma.

The diagnosis was leiomyosarcoma of the duodenum with ulceration and gastrointestinal hemorrhage. Accessory findings of hypertensive cardiovascular disease and a small hemangioma of the spleen were disclosed.

Case 2. A 60-year-old Negro woman was admitted to the Grady Memorial Hospital on April 6, 1956, with a chief complaint of indigestion and weakness. The patient's present illness began 6 months before admission with the onset of mild indigestion and tarry stools. A hemoglobin determination in the outpatient clinic was 6.0 gm. per cent. She denied hematemesis.

The patient had been treated for pulmonary tuberculosis in 1951 with good response. Diabetes mellitus was discovered on routine examination in 1951 and has been well controlled with diet and insulin therapy.

On admission, the pulse rate was 140; respirations, 24; blood pressure, 140/75. The patient was a well developed, 115-pound woman in no distress. Examination of the neck disclosed a diffuse enlargement of the thyroid. The chest was negative. The abdomen was soft with the liver palpable 1.5 cm. below the right costal margin. Rectal examination revealed a tarry, black stool. The remainder of the examination was negative.

Liver function studies were within normal range. A barium enema was negative. An upper gastrointestinal series revealed a bizarre configuration of the third portion of the duodenum in association with a mass.

The patient required 1500 cc. of whole blood to elevate the hemoglobin to 11.0 gm. per cent. She continued to have tarry bowel movements while on an ulcer regimen of antispasmodics, alkali and an ulcer diet. On the 13th hospital day abdominal exploration was performed.

Examination of the liver disclosed multiple nodules, some pedunculated, measuring up to 2 and 3 cm. in diameter. Located on the inferior border of the third portion of the duodenum was a 6 by 5 by 5-cm. mass (fig. 5). The lesion did not involve the head of the pancreas. It was approximately 10 cm. distal to Vater's ampulla. A palliative resection of the third portion of the duodenum was performed with an end-to-end anastomosis. A pedunculated nodule was removed from the surface of the right lobe of the liver.

The duodenal lesion was rubbery in consistency. Section revealed a pedunculated portion in the lumen of the duodenal segment with a sinus tract into the extraluminal mass (fig. 6). The nodule removed from the liver was firm and fibrous in consistency. Histologic examination disclosed whorls of spindle shaped cells. Anaplastic features were noted in the nuclei. The tumor extended

FIG. 5. Leiomyosarcoma of the third portion of the duodenum delivered anteriorly after having freed the hepatic flexure of the colon (case 2).

FIG. 6. The serosal view of the opened duodenum (shown in fig. 5) with the large ulcerated portion being intralumenal (pointer) and the smaller extralumenal portion being subserosal.

into the submucosa of the duodenum. Mitoses were present.

The diagnosis was leiomyosarcoma of the duodenum, with sinus tract formation, and multiple metastasis to the liver.

The patient did well postoperatively, taking a full diet on the eighth postoperative day. Her course was complicated by a wound infection that later required a secondary closure. She is now being followed in the outpatient clinic, 3 months after resection, with no symptoms referable to the gastrointestinal tract.

DISCUSSION

Massive gastrointestinal hemorrhage such as presented in the first patient constitutes a serious problem. The more frequent causes of massive bleeding were considered, but the diagnosis of duodenal tumor was not entertained. The lack of x-ray findings and the cardiac status in the first patient contributed to the failure of management. With positive clinical and x-ray findings, and previous experience with a duodenal tumor, a preoperative diagnosis of duodenal tumor, probably leiomyosarcoma, was made in the second patient.

Leiomyosarcoma originates in the muscular coats of the bowel. It is possible that benign leiomyomas may undergo malignant changes, but all pathologists do not agree on this point. McFarland[18] and others believe that the presence of a metastatic lesion alone offers proof of malignancy. Benign or malignant lesions may be entirely submucosal, entirely subserosal, or a combination of both types with the formation of "hourglass" or "dumbbell" lesions. Bleeding in the submucosal lesions results from central necrosis, liquefaction, and the formation of pseudodiverticula. Marshall and Cherry[15] state that it is usually the subserosal lesion that leads to exsanguinating hemorrhage.

A review of the literature revealed 39 previously reported cases (table 1). The duration of symptomatology varied from "days" to 13 years, supporting the belief that these are slow growing tumors. Evidence of gastrointestinal bleeding was the most frequent finding, occurring in 25 of 41 patients. Of these, 8 had fairly massive bleeding. Weight loss was noted in 20 patients. Abdominal pain, noted in 19 cases, was the primary complaint in 10 cases; the description being similar to that of peptic ulcer pain.

Physical examination disclosed palpable abdominal masses of varying sizes and mobility in 18 patients. These tumors were demonstrated to vary from 1.5 cm. to the size of a "fist." Jaundice was an infrequent finding, occurring in 3 patients with lesions in the second portion of the duodenum. One patient had jaundice with a lesion in the first portion of the duodenum, but the lesion communicated with an abscess that compressed the common duct.

Surgical resection of the lesion was performed in 20 of 26 patients who were submitted to surgery. Procedures varied from simple excision to pancreaticoduodenectomy, right colectomy with choledochojejunostomy, pancreaticojejunostomy, gastrojejunostomy and ileotransversostomy. Metastatic lesions were observed in 12 patients, the liver being most frequently involved.

Owing to the anatomical position of the duodenum, these tumors may cause complications by encroachment on adjacent structures, or they may remain relatively asymptomatic. Obstruction of the duodenum has not been reported, although 4 patients had intraluminal polypoid tumors. Other complications included perforation and fistula formation; 2 patients had rupture of the duodenal tumors with resulting

TABLE 1

Summary of duodenal leiomyosarcomas

No.	Author	Sex	Age	Duration of Symptoms	Gastrointestinal Bleeding	Pain	Weight Loss	Palpable Mass	Size	Duodenal Site	X-Ray	Operation	Results
									cm.				
1	Wesener[33]	M	55	8 mo.	+		+	+	7	2			Death
2	Ghon and Hintz[7]	M	81					+	15				Death
3	Von Salis[31]	M	40	7 yr.	−			+	15	4		Laparotomy	Death
4	Kathe[13]	M	57							4			
5	Brdiczka[3]	F	60	2 wk.	+	++		+	15	1	+	Resection	Death
6	Andersen and Doob[1]	M	37	5 yr.	+++	+	+	+	18	3	−		Death
7	Silverstone[28]	F	51	2 yr.		++	+	+	5	3	−	Resection	Death
8	Stommel[29]	M	39	5 wk.		++	+	+	"Fist"	2		Laparotomy	Death
9	McFarland[18]			13 wk.									
10	Seymour and Gould[26]	M	54	3 mo.	−	++	+	+	1.5	1	+		Death
11	Nickerson and Williams[21]	F	70	2 mo.		++				2			Death
12	Nickerson and Williams[21]	M	65	2 yr.		++	+		15	2			Death
13	Mendillo and Koufman[20]	M	49	3 yr.	+	+	+	−	10	2	+	Resection	Well
14	Foshee and McBride[6]	M	63	3 mo.			+	+	10	3	−	Resection	Death
15	Harrington and Ganshorn[8]	F	38	8 mo.	++	−		−	4	3	+	Resection	Well
16	Itikawa[11]	M	42	13 yr.	+	+	+	+	"Fist"	1		Laparotomy	Well
17	Mayo[16]												
18	Mayo[16]												
19	Henning and Garland[9]	F	65	3 mo.	+++	−	+	+	8	3	+	Resection	Death
20	Shackleford *et al.*[27]	F	61	3 mo.	++				8	2	+	Resection	Death
21	McCullough[17]	F	42	"mos."	+	++	+	+	8	3			Death
22	Williams[34]	F	30	"days"	+	++		−	4	4		Laparotomy	Death
23	Brunschwig and Tiholiz[4]	M	45	1 mo.	++				11	3	+	Resection	Well
24	Nitshe and Suckle[22]	F	64	6 mo.	++				2	2	−		Death
25	McLean[19]	M	50	4 mo.		−	+	−	6	1, 2	−	Laparotomy	Death
26	Heymann and Clark[10]	F	42	2 yr.	++		+	+	5	2	+	Resection	Death
27	Schwartz *et al.*[25]	F	55		+++	+		−	7	2	−	Resection	Well
28	Swartz and Eckman[30]	M	56	5 mo.	++		+	−	2.5	2		Resection	Well
29	Ripstein and Flint[24]	M	44	5 yr.	+++	+		−	2	2	−	Resection	Death
30	Ripstein and Flint[24]	M	45	5 mo.	+	++	+	+	15	1, 2		Resection	Death
31	Ripstein and Flint[24]	F	53	3 yr.	+	++		+		3		Resection	Death
32	Ayers and Cunningham[2]	F	80	3 yr.	++		+		3	2		Resection	Well
33	Weinstein and Roberts[32]	M	54	6 mo.	++	+	+	+	20	3	+	Resection	Well
34	Richardson and Flatt[23]	F	47	3 yr.	+++			+		2	−	Resection	Well
35	Richardson and Flatt[23]												Death
36	Zintel[35]	M	44	8 mo.	++		+	+		4	+	Resection	Death
37	Elias *et al.*[5]												Death
38	Judd and Hill[12]	M	31	5 mo.	+++					2		Resection	
39	Lopez[14]	F	60	1 yr.		+	+		2	2		Laparotomy	Death
40	Amerson	F	60	6 mo.	++	+		−	6	3	+	Resection	Well
41	Lumpkin	M	65	1 yr.	+++	+	+	−	4	2	−		Death

peritonitis and death; and 6 had duodenal fistula formation which further complicated the problem.

In summarizing the cases reported, x-ray examinations were helpful in 11 patients and of no aid in 9; 26 patients were dead at the time of their respective case report in the literature and 10 were known to be living. This would represent a high mortality rate of 72 per cent. This high mortality is due not to the malignant nature of the tumor but to the complications of surgery in the duodenal region, such as anastomotic breakdown, and duodenal and pancreatic fistula formation.

Since the tumor is radioresistant, surgical therapy offers the only benefit. Even in the presence of rather extensive metastatic disease, the primary tumor should be resected if possible to prevent hemorrhage, perforation, or possible obstruction. With increasing knowledge of surgical procedures in the area of the duodenum, complications of surgery itself should not be the underlying cause of poor results. The surgery may consist of only simple excision or may necessitate block resections with portions of the pancreas and colon, depending upon the site and attachment of the tumor.

SUMMARY

A review of the literature discloses 39 previously reported patients with leiomyosarcoma of the duodenum. Case reports of 2 recent patients with severe gastrointestinal hemorrhage are presented. The symptoms of both patients were suggestive of peptic ulcer. The death of 1 of the patients was caused by massive bleeding. The other patient had a palliative resection of the duodenal tumor and is at present doing well, without symptoms referable to the gastrointestinal tract.

When massive hemorrhage is accompanied by an intralumenal mass of the duodenum, leiomyosarcoma should be considered. Early surgical resection is the treatment of choice.

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REFERENCES

1. Andersen, D. H., and Doob, E. F.: Leiomyosarcoma of the duodenum. Arch. Path., *16:* 795, 1933.
2. Ayers, W. B., and Cunningham, G. A.: Leiomyosarcoma of the duodenum with gastrointestinal hemorrhage. Surgery, *31:* 922, 1952.
3. Brdiczka, I. G.: Die Tumoren der Bulbus duodeni und ihre röntgenologishe Diagnose. Röntgenpraxis, *3:* 625, 1931.
4. Brunschwig, A., and Tiholiz, I. C.: Surgical treatment of malignant tumors of the duodenum exclusive of those arising from papilla of Vater. S. Clin. North America, *26:* 163, 1946.
5. Elias, W. S., Lund, C. C., and Yonemoto, R.: Neoplasms of small intestine. Am. J. Surg., *88:* 384, 1954.
6. Foshee, J. C., and McBr[illegible], W. P. L.: Leiomyosarcoma of the duodenum. Report of a case and review of the literature. J. A. M. A., *112:* 2497, 1939.
7. Ghon, A., and Hintz, A.: Über maligne Leiomyome des Intestinaltraktes. Beitr. path. Anat., *45:* 89, 1909.
8. Harrington, S. W., and Ganshorn, J. A.: Leiomyosarcoma of the third portion of the duodenum. Report of a case. Proc. Staff Meet., Mayo Clin., *15:* 74, 1940.
9. Henning, B. H., and Garland, L. H.: Leiomyosarcoma of the duodenum. Radiology, *37:* 353, 1941.
10. Heymann, J. A., and Clark, G. G.: Leiomyosarcoma of duodenum. Am. J. Surg., *80:* 119, 1950.
11. Itikawa, H.: Falle von Leimyom des Verdauungstraktus. (Leiomyosarcoma duodeni.) Arch. Jap. Chir., *17:* 1270, 1940. Cited by G. W. Horsley and R. L. Means: Leiomyosarcoma of the jejunum, with two case reports and a review of the literature. Ann. Surg., *141:* 799, 1955.
12. Judd, C. S., Jr., and Hill, R. L.: Pancreatoduodenectomy for Leiomyosarcoma of the duodenum. Ann. Surg., *139:* 103, 1954.
13. Kathe, H.: Zur kenntnis des myoblastiochen Sarkoms. Virchows Arch. path. Anat., *187:* 265, 1907.
14. Lopez, J. F.: Leiomyosarcoma of the duodenum. Report of a case with necropsy. Gastroenterology, *30:* 123, 1956.
15. Marshall, S. F., and Cherry, J. W.: Smooth muscle tumors of the alimentary canal. S. Clin. North America, *35:* 719, 1955.
16. Mayo, C. W.: Malignancy of the small intestine. West. J. Surg., *48:* 403, 1940.
17. McCullough, K.: Leiomyosarcoma of the duodenum. New York J. Med., *44:* 1248, 1944.
18. McFarland, J.: Malignant myoma. Am. J. Cancer, *25:* 530, 1935.
19. McLean, R. B.: Malignancies of the duodenum. South. Surgeon, *14:* 430, 1948.
20. Mendillo, A., and Koufman, W. B.: Diverticulum and sarcoma of the duodenum. Report of a case. New England J. Med., *219:* 432, 1938.
21. Nickerson, D. A., and Williams, R. H.: Malignant tumors of the small intestine. Am. J. Path., *13:* 53, 1937.
22. Nitshe, G. A., Jr., and Suckle, H. M.: Leiomyosarcoma of ampulla of Vater associated with tumor of eighth nerve and neurogenic perforation of duodenum; Report of a case and review of literature. Am. J. Clin. Path., *17:* 827, 1947.
23. Richardson, J. E., and Flatt, A. E.: Unusual

small-intestinal causes of anemia. Brit. M. J., *2:* 311, 1953.

24. Ripstein, C. B., and Flint, G. W.: Leiomyosarcoma of gastrointestinal tract. Gastroenterology, *20:* 315, 1952.
25. Schwartz, N. H., Swingle, R. C., and Raymond, E. A.: Malignant tumors of the duodenum. Report of 2 cases. A. M. A. Arch. Int. Med., *87:* 410, 1951.
26. Seymour, W. J., and Gould, S. E.: Leiomyosarcoma of the duodenum. Report of a case. Am. J. Cancer, *28:* 572, 1936.
27. Shackleford, R. T., Fisher, A. M., and Firor, W. B.: Duodenal tumor of unusual character. Ann. Surg., *116:* 864, 1942.
28. Silverstone, M.: Sarcoma of the duodenum. Report of a case. Brit. J. Surg., *22:* 332, 1934.
29. Stommel, H. G.: Ein Fall von Leiomyosarcoma duodeni mit Lebermetastasen, Thesis, Bonn, Bonn University, 1935. Cited by G. W. Horsley and R. L. Means: Leiomyosarcoma of the jejunum, with two case reports and a review of the literature. Ann. Surg., *141:* 799, 1955.
30. Swartz, W. T., and Eckman, W. G., Jr.: Case report of leiomyosarcoma of the duodenum. Surgery, *29:* 581, 1951.
31. Von Salis, H. W.: Über das Sarkom des Duodenum insbesondere das myosarkom. Deutsche Ztschr. f. Chir., *160:* 180, 1920.
32. Weinstein, M., and Roberts, M.: Leiomyosarcoma of the duodenum. A. M. A. Arch. Surg., *66:* 318, 1953.
33. Wesener, F.: Ueber ein telangectatisches myom des duodenum von ungewöhnlicher Grösse. Arch. F. Path. Anat., *93:* 377, 1883.
34. Williams, M. H. L.: A case of perforating leiomyosarcoma of the duodenum associated with parturition. M. J. Australia, *1:* 585, 1944.
35. Zintel, H. A.: Leiomyosarcoma of the duodenum. A. M. A. Arch. Surg., *69:* 612, 1954.

THE MANAGEMENT OF ESOPHAGEAL HIATAL HERNIA COMPLICATED BY A LOWER ESOPHAGEAL RING*

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Esophageal hiatal hernia complicated by an acquired lower esophageal stricture is a clinical entity that has been described on many occasions in the past; however, the surgical observations in cases of esophageal hiatal hernia with lower esophageal ring have been reported by a limited number of authors.[2, 7]

We believe that lower esophageal stricture and the lower esophageal ring are separate and distinct pathologic processes and should be managed by entirely different methods.

In 1953 Schatzki and Gary[5] and Ingelfinger and Kramer,[3] following independent investigations, described a narrow circular contraction that was noted to be present in the region of the cardioesophageal juncture. Ingelfinger and Kramer thought that the ring was formed as a muscular contraction wave approached the lower esophagus, whereas Schatzki and Gary described the ring as being a preformed fixed structure that was present at all times. Improved radiographic techniques[6] revealed that a lower contraction ring was present in about 5 per cent of those individuals undergoing an upper gastrointestinal tract survey. Multiple radiographic studies in individual patients revealed that the lower esophageal rings were constantly present and readily visualized. Investigative studies by Wolf,[8] consisting of the injection of iodized oil into the esophageal wall at the level of the cardioesophageal juncture, and autopsy studies by MacMahon and associates,[4] add to the accumulating evidence that the lower esophageal ring is located at the exact cardioesophageal mucosal juncture. An esophageal hiatus hernia of the sliding type has usually been reported as a concomitant finding in those patients with symptomatic lower esophageal rings.

MATERIAL STUDIED

In a group of 132 patients operated upon because of a symptomatic diaphragmatic hernia, only 4 were found to have an associated lower esophageal ring. The first patient was a 35-year-old white man with a history of increasing dysphagia, particularly noted after the ingestion of solid foods, for a period of 8 months before surgery. For a 3-year period before the onset of dysphagia, this patient noted severe attacks of regurgitant esophagitis; however, as dysphagia appeared the esophagitis decreased and was noted to be absent for a 6-month period before surgery. X-ray examination (fig. 1) of this patient's lower esophagus revealed a 3-mm., circular ring that was noted to be constantly present with multiple x-ray examinations. There was an associated sliding esophageal hiatal hernia of moderate size. The second patient was a 52-year-old white man with a history of persistent epigastric and substernal burning pain over an 8 to 9 year period. Dysphagia, after swallowing solid foods, first appeared 18 months before the operative procedure and was noted to be progressive until only liquids and semisolids could be swallowed. Radiographic studies (fig. 2) revealed a small sliding hiatal hernia, and at the cardioesophageal juncture there was noted a constant lower esophageal ring deformity that was 3 to 5 mm. long with an estimated internal diameter of 6 to 8 mm. The third and fourth patients (figs. 3 and 4) were white men, 36 and 59 years of age. They both related histories of steady progressive dysphagia of 3 years' duration and difficulty in swallowing was noted primarily following the ingestion of solid foods. It was of interest to note that neither of these patients gave any history of epigastric distress or symptoms suggestive of regurgitant esophagitis.

In none of the 4 patients studied was there a history of traumatic injury to the esophagus in the form of a caustic substance, or instrumentation of the presence of an indwelling nasogastric tube. Except for the symptomatology noted, past history was noninformative in this group.

SURGICAL MANAGEMENT

In all 4 of the individuals studied, identical surgical procedures were utilized; left thoracot-

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FIG. 1. Lower esophageal ring 3 mm. in length. Associated diaphragmatic hernia not demonstrated with this view.

FIG. 2. Lower esophageal ring with hiatal hernia visible. The diaphragm-like structure was 4 mm. in thickness with a 6- to 8-mm. lumen.

omy was performed. The lower esophagus and the herniated portion of the interthoracic stomach were mobilized. A counter incision was made in the diaphragm and after reduction of the herniated stomach an Allison[1] type repair of the esophageal hiatus hernia was performed. By utilizing the incision in the diaphragm, a gastrotomy was performed in the cardia of the stomach and with retraction, the cardioesophageal juncture was palpated and observed under direct vision. In 3 of the patients, palpation and visualization revealed a thin ring of tissue that was located at the juncture of the esophageal and gastric mucosa (fig. 5). In these 3 patients it was noted that the finger could be inserted, but that the lower esophageal ring

FIG. 3. Lower esophageal ring that appears as an unilateral indentation at cardioesophageal juncture.

FIG. 4. Belated indentation shows location of lower esophageal ring.

firmly encircled the palpating digit. After inserting 2 fingers into the rigid lower esophageal ring, stellate fractures appeared in the mucosa that formed the constricting band, as demonstrated by the pointer in figure 5. In one of the patients (fig. 6) the gross findings were noted to be quite different. Direct visual inspection revealed a definite diaphragm rather than a submucosal ring. The internal diameter of the residual esophageal lumen was estimated to be 5 to 7 mm. The diaphragm was dense, 3 to 5 mm. thick, and totally resisted attempts at digital fracture or laceration. In this particular case the diaphragm was excised at the point of its juncture with the internal wall of the esophagus by utilizing electric cautery.

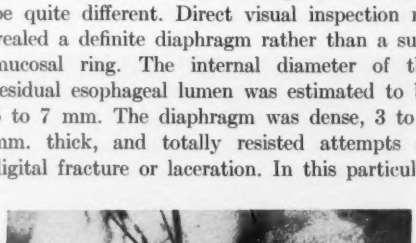

FIG. 5. Lower esophageal ring as viewed through the gastrotomy incision. Pointer locates a laceration in the fibrous ring following avulsion of the lower esophageal ring.

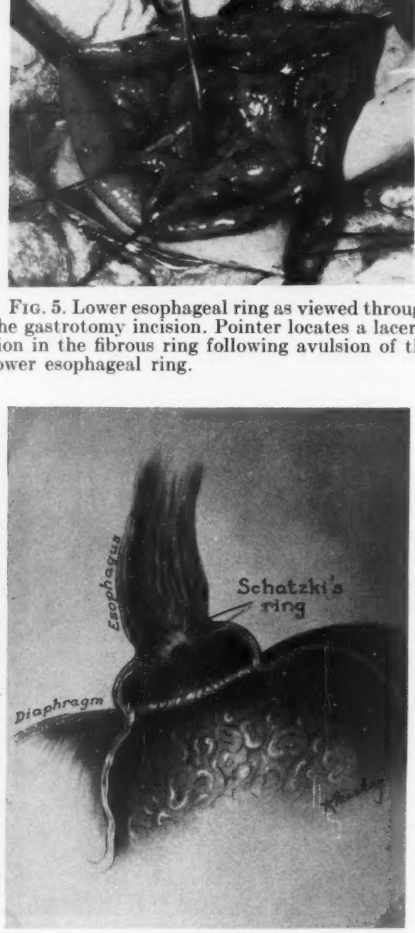

FIG. 6. Artist's concept of the diaphragm-like ring with an associated hiatal hernia.

FIG. 7. Postoperative study demonstrating repair hiatal hernia. The lower esophageal ring has been indicated.

RESPONSE TO OPERATIVE MANAGEMENT

Of the 4 patients presented, 3 made an uneventful recovery from the surgical procedure and have remained free of symptoms. Postoperative x-ray study of the lower esophagus in each patient demonstrated repair of the hiatal herniation and the absence of any indication of a residual lower esophageal ring. Figure 7 is the postoperative examination of the first patient studied (fig. 1). There is no terminal esophageal dilation and the ring-like constriction at the cardioesophageal juncture cannot be demonstrated.

The second patient that was presented (figs. 2 to 5) died suddenly on the 10th postoperative day and autopsy revealed a massive pulmonary embolus in the left pulmonary artery. Inspection of the cardioesophageal area demonstrated satisfactory repair of the hiatal hernia and there was no residual obstruction in the region of the diaphragm-like structure that was resected at the time of the operative procedure.

DISCUSSION

The purpose of this paper is to present the preoperative studies and the observations at the time of surgical repair in the cases of 4 pa-

tients with esophageal hiatal hernia complicated by a lower esophageal ring. Although considerable controversy exists regarding the etiology, location, and clinical significance of the lower esophageal ring, we feel, after having visualized and palpated the ring-like structure in 4 individual patients, that the lower esophageal ring is a distinct pathologic entity and that it becomes highly symptomatic and requires surgical correction when it is associated with a sliding type of diaphragmatic hernia. We have not observed any patients with symptomatic lower esophageal ring that have not also presented a concomitant sliding esophageal hiatal hernia. It appears from our clinical study that in some patients the lower esophageal ring becomes a protective mechanism in the prevention of regurgitant esophagitis. In 2 patients a history of severe regurgitant esophagitis with onset of dysphagia from the lower esophageal ring was reported, and the degree of esophagitis decreased and finally disappeared. The problem of whether or not an operative management of the lower esophageal ring should be employed has been considered. With a concomitant hiatal hernia it seems to us that the best method of management is a satisfactory repair of the existing herniation and direct visual observation of the area of constriction at the cardioesophageal juncture with subsequent dilation and avulsion of the constricting band or resection of a fibrotic type of lower esophageal diaphragm.

Preoperative esophagoscopy was performed as a preoperative study in all 4 of the patients presented. In 3 of the patients studied the esophagoscope was passed through the cardioesophageal juncture and we were unable to identify a lower esophageal ring. In the patient with the obstructing lower esophageal diaphragm, the operator was unable to pass the esophagoscope through the cardioesophageal juncture; however, the true nature of the existing pathology was not demonstrable until the lesion was exposed at the time of gastrotomy.

SUMMARY

During the performance of upper gastrointestinal x-ray study, lower esophageal rings will be demonstrated in about 5 per cent of the patients studied; however, it seems that a concomitant esophageal hernia must be present to cause the lower esophageal constrictions to become symptomatic.

The lower esphageal ring is a different pathologic process as compared to the lower esophageal stricture and requires a different and, fortunately, a less complex operative procedure.

Experience with four cases indicates that a satisfactory method of management is that of complete repair of the diaphragmatic hiatal hernia and under direct vision avulsion of the thin mucosal constriction ring or resection of the more dense and fibrous diaphragm.

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REFERENCES

1. Allison, P. R.: Reflux esophagitis, sliding hernia and the anatomy of repair. Surg. Gynec. & Obst., *92:* 419, 1951.
2. Bugden, W. F., and Delmonico, J. E., Jr.: Lower esophageal web. J. Thoracic Surg., *31:* 1, 1956.
3. Ingelfinger, F. J., and Kramer, P.: Dysphagia produced by contractile ring in lower esophagus. Gastroenterology, *23:* 410, 1953.
4. MacMahon, H. E., Schatzki, R., and Gary, J. E.: Pathology of lower esophageal ring. Report of a case with autopsy, observed for nine years. New England J. Med., *259:* 1, 1958.
5. Schatzki, R., and Gary, J. E.: Dysphagia due to diaphragm-like localized narrowing in lower esophagus ("lower esophaegeal ring"). Am. J. Roentgenol., *70:* 911, 1953.
6. Schatzki, R., and Gary, J. E.: The lower esophageal ring. Am. J. Roentgenol., *75:* 246, 1956.
7. Spencer, J. R., Hammer, R. W., and Wierman, W. H.: Lower esophageal rings: report of fourteen cases. Am. Surgeon, *25:* 20, 1959.
8. Wolf, B. S.: Motor phenomena in distal esophagus: diagnosis of minimal hiatal herniation. Exhibit. Presented at 58th annual meeting of American Roentgen Ray Society, Washington, D. C., October 1 to 4, 1957.

CHANGING PATTERNS RELATIVE TO THE SURGICAL TREATMENT OF PULMONARY ARTERIOVENOUS FISTULAS*

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During the past 14 years the authors have had the opportunity to study and treat 12 patients with pulmonary arteriovenous fistulas. This lesion has been considered a clinical rarity. Bosher and associates[2] recently added 98 previously unpublished cases by means of correspondence with 67 contributors, thus increasing the published cases reported to 350. It is not our plan here to review the historic background relative to this disease or the literature pertaining thereto. In a recent report relative to various types of pulmonary vascular fistulas and anomalies,[1] we have summarized this matter in detail. We also recommended a new classification for lesions in this category and subdivided congenital pulmonary arteriovenous fistulas of the usual type into two subdivisions. The first consists of the solitary saccular form of the disease with one or at most two feeding arteries. The second subdivision includes the racemose type with multiple fistulas and feeding vessels. In our experience a different clinical picture is presented by these two different types of lesions. The simple saccular form tends not to present evidence of polycythemia or clubbing of the fingers and toes. The racemose type of lesion characteristically presents these phenomena.

Several excellent reviews of the literature are available and the reader is referred to the articles by Yater and co-workers[9] and Quemerch,[4] both of which appeared in 1949; Muri[5, 6] presented valuable reviews in 1953 and 1957. Other valuable reviews of the subject were presented in 1953 by Sloan and Cooley[7] and Stringer and associates.[8]

The main reason for our presentation at this time is to add 12 new patients to the literature and to discuss the progressive trend in the surgical treatment of this disease toward local excision of the lesion.

The lesions to be considered herein include only those cases in which there is an abnormal congenital communication between the pulmonary arteries and the pulmonary veins. Such lesions may be single or multiple and have been reported to occur in all lobes of the lung. Bosher and associates[2] described 5 patients in whom all five lobes of the lung were involved with multiple fistulas. It is interesting that 2 of our 12 patients presented this same syndrome. Review of the literature indicates that 35.6 per cent of patients have multiple fistulas, whereas 64.4 per cent appear to have single lesions. There does not seem to be any significant predilection for the disease to involve one area. The fact that 55 per cent of the single fistulas occur in the lower lobes hardly more than parallels the total percentage of volume of pulmonary tissue made up by these areas of the lung. Of patients with pulmonary arteriovenous fistulas, 44 per cent also demonstrate evidence of generalized hereditary telangiectasia (Rendu-Osler-Weber's disease).

The pulmonary lesions show a wide variation in size and conformation. The degree of right-to-left shunting of the blood also varies markedly. There seems to be a significant tendency for these lesions to be located almost totally or partially in a subpleural plane. However, some are located deep in the lung tissue and may be difficult to palpate.

Microscopic study usually reveals an abrupt transition from a normal arterial wall to either a capillary or aneurysmal type of wall. The sac may be of variable thickness, single or multiple, or multiloculated. The wall of the sac tends to be made up of elastic tissue fibers, shadow forms of smooth muscle cells and built up mainly by layers of hyalinized fibrous tissue. The aneurysmal sac may be found to contain thrombi. The draining veins may be single or multiple and tend to be enlarged and tortuous. Thickening, or so-called "arterialization," of the vein wall is common.

The fundamental pathophysiology consists of

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varying degrees of shunting of blood in the right-to-left direction. Such shunts may be negligible and the arterial oxygen desaturation may be minimal or apparently absent. Severe degrees of desaturation as low as 64 per cent have been noted. Except with massive fistulas there are no secondary effects upon the heart. With the presence of universal fistulas involving all lobes or massive more localized fistulas, cardiac failure particularly with right ventricular hypertrophy has been noted.

The clinical findings in these patients may be variable. Large shunts, particularly with the racemose type fistula, are prone to produce polycythemia and clubbing. Telangiectasia is seen in those patients having Rendu-Osler-Weber syndrome.

The clinical symptoms may be minimal to absent. The most common symptom is dyspnea. Hemoptysis occurs fairly frequently. Another hemorrhagic manifestation may be hemothorax. The cerebral complications are frequent and varied. With massive shunts and severe polycythemia, one may expect headache, thickness of speech, vertigo, episodes of unconsciousness, convulsions or frank strokes. Cerebral manifestations may be seen in any type of fistula when the pulmonary arteriovenous aneurysmal sac contains thrombi or vegetations. A moderate number of patients with pulmonary arteriovenous fistulas have had fatal hemorrhagic episodes either in the form of hemoptysis or hemothorax.

The usual laboratory data may be within normal limits or reveal varying degrees of polycythemia. Such a polycythemia involves an increase in red cell mass and of total blood volume without concomitant increase in plasma volume. Of importance to the surgeon is the tendency of patients with severe cyanosis to develop thrombocytopenia. The white cell count is normal.

Roentgenograms provide the most important and consistent signs of this disease. They present an opacity which may be round or lobulated, connected to the major pulmonary vascular trunks by broad band shaped vascular shadows. The lesions may be single or multiple. Fluoroscopy may be of assistance in the study of these lesions. Laminagram may outline a feeding vessel not visible on plain roentgenograms and thus direct the clinician to the need for contrast studies. Angiocardiography is an absolute essential for proving the diagnosis of pulmonary arteriovenous fistula. It is also considered a mandatory integral part of the management of every patient with this disease because of the inherent tendency to multiplicity of these lesions.

The differential diagnosis involves possible consideration of other types of congenital cardiovascular abnormalities, pulmonary granuloma or neoplasm, and occasionally polycythemia vera.

The natural course of the disease is usually one of slow progression. Sudden termination due to cerebral complications such as embolism, cerebral abscess or cerebral thrombosis may occur. Massive hemorrhage is probably more frequent than previously considered. Doughtry[3] described an asymptomatic saccular type of fistula in a young pregnant woman. Surgery was recommended but refused. The patient died 4 weeks later enroute to the hospital, due to sudden massive hemoptysis.

REVIEW OF PERSONAL CASES

Since 1946 we have had opportunity to see 12 patients with what we classify as congenital (pure type) pulmonary arteriovenous fistula. Of these, 5 patients fall into the subcategory of solitary sacculation with a single feeding artery. The remaining 7 patients presented with the racemose type of lesion with multiple feeding arteries.

Congenital (Pure Type) Pulmonary Arteriovenous Fistula

Saccular with single feeding artery. In this group there were 5 patients, 4 men and 1 woman. In no instance was polycythemia or clubbing encountered. In 3 patients a systolic or diphasic murmur was audible. Telangiectasia of the skin and mucous membranes was found in only 1 of this group. Angiographic studies, performed in 4 cases, revealed the solitary saccular type of lesion. In the fifth patient a large solitary oval mass was seen on screening x-ray studies (fig. 1). A suspicion of a feeding vessel was created by study of laminagraphic examinations. Angiocardiographic studies were not permitted by the patient's husband, who happened to be a radiologist! At the time of surgery a large saccular lesion was found occupying the right middle lobe. No other demonstrable lesions were seen in the right pleural cavity. Following surgery the excised lobe was studied radiologically with the

FIG. 1. Patient R. M. G. The posteroanterior roentgenogram revealed a large oval shadow in the right midlung field. Laminagrams raised the question of possible pulmonary arteriovenous aneurysm but angiographic studies were not permitted.

aid of injection of contrast media into the pulmonary arteries; a second unsuspected fistula was revealed (fig. 2). Subsequent radiographic observation of this patient during the ensuing 9 years has failed to reveal any evidence of other fistulas.

Of the patients presenting with single saccular lesions, 2 happened to be brothers; 2 other siblings in this family are known to have Rendu-Osler-Weber syndrome. The first of these brothers was initially admitted because of coma and hemiparesis. X-ray examination revealed a mass in the right lower lobe area. When the patient recovered from his stroke further x-ray study suggested the lesion might be a fistula and this was proved by angiocardiography (fig. 3). The internist considered the stroke to be embolic from the fistula and that it was indication *per se* for operative correction of the pulmonary lesion. A total right lower lobectomy was performed. The specimen on examination revealed a single sac 3 cm. in diameter with a single feeding artery. Two moderately large and tortuous veins draining the lesion were found. The sac contained a laminated, organized blood clot with surface changes compatible with the suggestion that it had been a source of the cerebral embolus. Follow-up during the ensuing 8 years revealed no recurrence of cerebral difficulty, and the patient appears clinically cured.

The 5 patients in this group were treated by the following methods. One patient had a total pneumonectomy in 1946; the dissection of this lung suggested to us that this was an unnecessary sacrifice of useful lung tissue. The next 2 patients were treated by right lower and right middle lobectomy, respectively. In October 1953, a solitary peripheral lesion 4 cm. in diameter was surgically treated by enucleation without loss of adjacent healthy lung tissue. This operative procedure was performed by the subsegmental enucleation technique and did not require occlusion of the blood supply to this left upper lobe during the procedure. One subsequent patient with a large, somewhat deeply positioned saccular lesion was treated by segmental resection in October 1958.

Follow-up of these patients for a period of 5 months to 13 years has shown no evidence of recurrence of disease or symptomatology.

Racemose type arteriovenous fistula with multiple feeding arteries. Of the 7 patients presenting with this type of lesion, polycythemia was present in 6 patients. In the 1 patient not presenting polycythemia there was a strong suggestion that such a factor had been present. This patient was first seen as an emergency because of shocklike state. Examination revealed a massive left hemothorax. Hematologic studies made at that time revealed a red blood cell count of 5.4 million with a hemoglobin of 7.9 gm. and an hematocrit of 30 mm. The patient did present some evidence of clubbing but no evident cyanosis. No murmur was audible. Initial therapy consisted of multiple thoracentesis, with immediate satisfactory clinical response. Transfusions were given. Following removal of the blood from the left pleural space a mass was visible. The initial impression was that we were probably dealing with a neoplasm with secondary hemothorax. No tendency for the hemothorax to recur was noted. A few months later the patient was admitted for detailed study. Laminagraphy did not show any definite feeding vessels to the lesion. Plain roentgenograms (fig. 4) suggested a peripheral mass of undetermined origin. Surgical exploration was performed revealing a multiloculated lesion located subpleurally and the entire lesion was quite superficial in position. Localized excision could be performed without difficulty by initial ligation of the feeding branches which were located

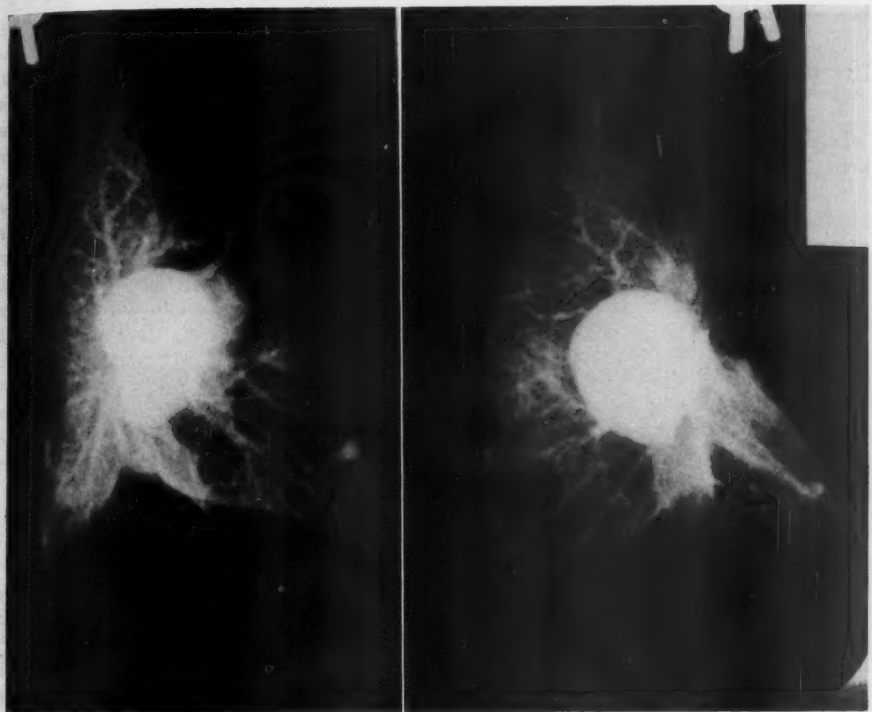

FIG. 2. Patient R. M. G. Roentgenogram of postoperative injection of Diodrast into right middle lobe arteries of specimen removed. Note large major aneurysm plus second small unsuspected pulmonary arteriovenous fistula not visible on plain roentgenograms.

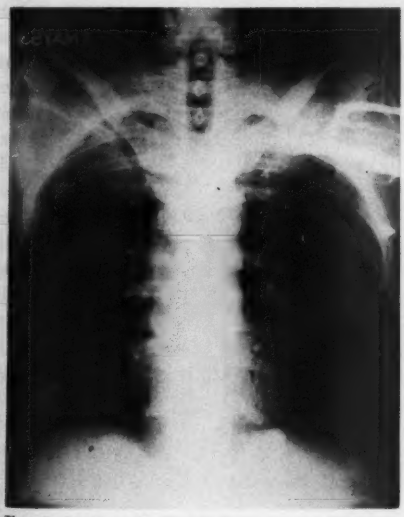

FIG. 3. Patient B. T. W. Pulmonary angiogram taken in anteroposterior position supine. Note lesion in right lower lung field. Area of absent contrast media in central portion might have been produced by laminated clot.

near the periphery of the lung. An uneventful recovery followed and the patient has had no difficulty in the subsequent 3 years of observation.

Another patient, age 43, was seen in February 1950. History revealed this white man had been moderately dyspneic and had fatigued easily throughout his life. Marked progression of symptoms with severe cyanosis had been noted for 15 years. Examination revealed angiomas of the lower lip and telangiectatic changes of the skin and mucous membranes. No murmur was audible. Fluoroscopy and chest roentgenograms (fig. 5) showed a cluster of lobulated densities occupying a large portion of the right lower lobe. This patient was of considerable interest because of the laboratory findings of a hemoglobin of 27 gm., a red blood cell count of 11 million and an hematocrit of above 75 mm. Measurements of blood volume showed a plasma volume of 1900 cc., a red cell mass of 6700 cc., with a total blood volume of 8600 cc. Operation consisted of a total right lower lobectomy be-

FIG. 4. Patient G. L. B. Posteroanterior roentgenogram taken 4 months after initial episode of spontaneous left hemothorax. Before exploration this patient was thought to have a neoplasm. Mass in upper lung field proved to be a superficially located racemose type of pulmonary arteriovenous aneurysm.

cause of the apparent almost total involvement of this lobe. Preoperative angiocardiogram had shown no evidence of other fistula formation. His postoperative course was complicated by a brief interval of cerebral thrombosis and transient left hemiplegia. This progressively improved leaving no residua. Postoperative studies, 5 months after surgery, showed that this total blood volume was 6700 cc., red cell mass 3140, and plasma volume 3560; 11 months after surgery the red blood cell count had dropped to 4.74 million, hemoglobin to 14.5 and the hematocrit to 46 mm. During the subsequent 8½

FIG. 5. Patient G. L. M. Pulmonary angiograms revealing a massive arteriovenous fistula of the racemose type occupying major portion of the right lower lobe.

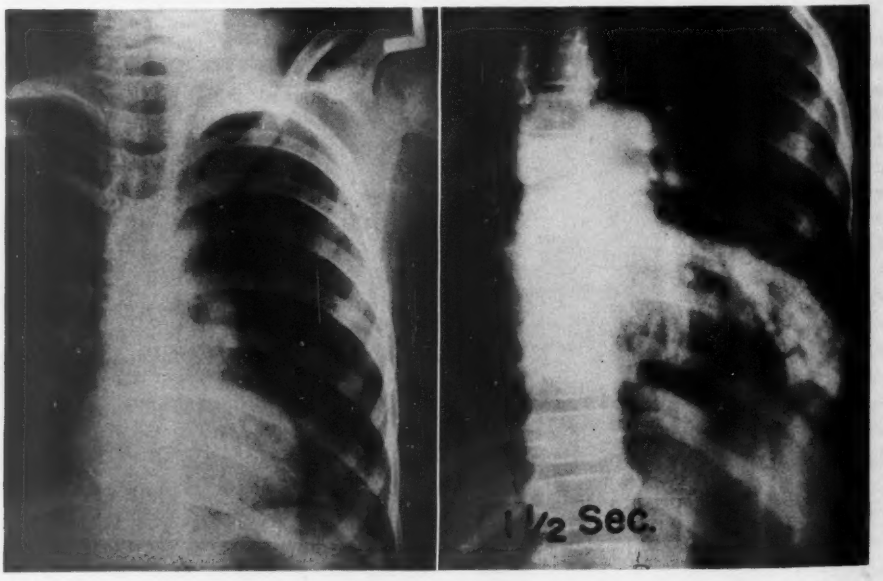

FIG. 6. Patient B. S. H. Pulmonary angiogram taken in a 7-year-old cyanotic female. This racemose type pulmonary arteriovenous fistula was cured by segmental resection (anteromedial basilar segment).

FIG. 7. Patient L. V. S. Racemose type pulmonary arteriovenous fistula demonstrated by pulmonary angiogram in a 43-year-old white man. No other lesions demonstrated. Treatment consisted of segmental resection.

years the patient has remained asymptomatic and fully employable.

Another patient of interest in this group is a 7-year-old girl who had been cyanotic since birth. A systolic murmur was heard over the area of the lingula and she was noted to have both cyanosis and clubbing. Angiocardiogram (fig. 6) revealed a 2 by 2-cm. lobulated arteriovenous fistula. At operation a racemose type of fistula with three feeding arteries occupying the basilar segment of the left lower lobe was found. This was treated successfully by segmental resection in March 1953, being one of the early segmental resections for this disease.

Of the other 4 patients in this group, 1 had a solitary racemose type of lesion (fig. 7), which was cured by segmental resection. Another patient with a solitary racemose type of lesion was found to have serious associated diseases; namely, hypertension, cerebral arteriosclerosis, generalized telangiectasia and his Bromsulphalein test relative to liver function revealed a 70 per cent retention. The patient showed evidence of weight loss, had recurrent syncope, repeated melena and confusion. Surgery was not recommended because of the associated other types of pathology. We received a report that he died at the age of 54, about 6 months after our study. The cause of death was not revealed.

Of the 7 patients with the racemose type of lesion, 2 were found to have universal pulmonary involvement. One of these was a 14-year-old colored girl, who had been cyanotic throughout her life. Severe progressive cardiac failure had been noted for several months before being seen by our group. Clubbing of the fingers and precordial systolic murmur were noted. The patient was severely polycythemic. She had suffered considerably from hemoptysis. Telangiectasia of the skin and mucous membranes was present. Roentgenographic studies revealed rather marked diffuse enlargement of the heart (fig. 8*A*). Angiocardiogram revealed multiple arteriovenous fistulas involving all lobes of both lungs (fig. 8*B*). Cardiac catheterization was performed revealing no evidence of shunts between either the auricles or ventricles. No surgery was advised. The patient died 7 months later, apparently from heart failure. Unfortunately, an autopsy could not be performed.

One other patient in this group suffered from universal pulmonary involvement (fig. 9). No surgery was recommended and the patient was discharged. We were notified several months later that this 24-year-old man had undergone a lingulectomy elsewhere in an attempt to remove the apparent largest conglomeration of fistulous

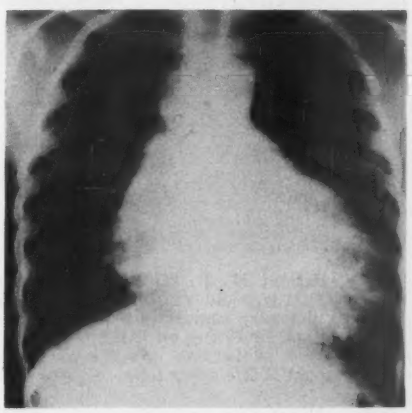

FIG. 8*A*. Patient W. M. C. Roentgenographic studies of a 14-year-old colored girl with severe cyanosis and heart failure. Teleroentgenogram shows massive cardiac enlargement and diffuse pulmonary infiltrates.

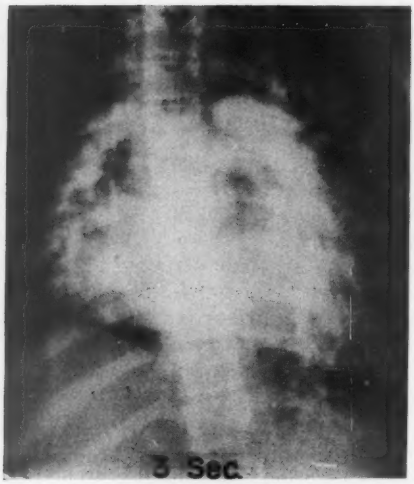

FIG. 8*B*. Patient W. M. C. Pulmonary angiogram proves presence of multiple arteriovenous fistula involving all five lobes of the lung.

FIG. 9. Patient M. B. H. Roentgenographic studies of a 24-year-old white man with severe cyanosis. Pulmonary angiogram proves lesions to be multiple and involving all five lobes. Largest conglomeration of fistulas appears to involve lingual segment of left upper lobe.

communications. The patient's death, on the eighth postoperative day, was apparently due to combined causes of cardiac failure and hydropneumothorax.

SUMMARY AND CONCLUSIONS

The authors have reviewed their experience with 12 personal cases presenting pulmonary arteriovenous fistulas. The lesions encountered fell into two basic groups: (1) congenital (pure type) pulmonary arteriovenous fistula consisting of a major solitary area of saccular lesion with a single feeding artery and one or two draining veins; and (2) congenital (pure type) pulmonary arteriovenous fistula of the racemose type composed of multiple fistulas and feeding arteries and veins. The 5 patients with the simple saccular form did not present evidence of associated polycythemia or clubbing. The racemose type of lesion presented these phenomena in all 7 patients with this type of pathology.

Brief reference has been made to the clinical picture presented. Some stress has been laid upon the frequency of hemoptysis. Such bleeding episodes may be fatal. Other patients may succumb from episodes of spontaneous hemothorax. The cerebral complications attendant to this disease are multiple.

Our initial patient was treated by total pneumonectomy. Study of the excised lung indicated that a much more conservative surgical approach to these lesions should be entertained. It is now our policy to utilize local excision without loss of adjacent lung tissue whenever possible. Occasionally, segmental resection or total lobectomy may be required. The most important single preoperative study consists of detailed pulmonary angiography in order to prove the lesion and to define the number of lesions present. Cerebral embolism in 1 case was considered to be a major indication for early surgical intervention.

Of the 12 patients, 2 presented universal pulmonary involvement and surgery was not recommended. In 1 patient, with a solitary racemose type of lesion, surgery was contraindicated because of severe associated hypertension, cerebral arteriosclerosis and cirrhosis. The remaining 9 patients were treated by surgical excision. Postoperative complications consisted of a mild cerebral thrombosis in 1 patient and potsoperative atelectasis in 1 other. Follow-up studies from 5 months to 13 years have revealed no evidence of recurrence of clinical symptoms or pathology in any of the 9 patients who underwent surgical resection. There have been no

surgical mortalities in these 9 personally operated cases.

It happened that none of our patients had any difficulty with thrombocytopenia. All cyanotic patients must undergo thorough preoperative study in this regard. Should such a complication present itself, it can be satisfactorily controlled by initial administration of pure fresh platelet infusions at the start of surgery. Repeated amounts can be given as needed during the operation.

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REFERENCES

1. Abbott, O. A., Haebich, A. T., and Van Fleit, W. E.: Considerations relative to classification and treatment of pulmonary vascular fistulae of all types. Cardiology, in press.
2. Bosher, L. H., Jr., Blake, D. A., and Byrd, B. R.: An analysis of the pathological anatomy of pulmonary arteriovenous aneurysms with particular reference to the applicability of local excision. Surgery, *45:* 91, 1959.
3. Doughtry, D.: Presented at the Southern Sectional Meeting of the American College of Chest Physicians, 1957.
4. Quemerch, R.: Contribution à l'étude des anéurysmes pulmonaire artério-veineux. Thèse de Lyon, 1949.
5. Muri, J. W.: Arterio-venous aneurysm of the lung. Dis. Chest., *24:* 49, 1953.
6. Muri, J. W.: Pulmonary arteriovenous aneurysm. In *Bronchopulmonary Diseases*, edited by E. A. Naclerio, Ch. 32. Paul B. Hoeber, Inc., New York, 1957.
7. Sloan, R. D., and Cooley, R. N.: Congenital pulmonary arteriovenous aneurysm. Am. J. Roentgenol., *70:* 183, 1953.
8. Stringer, C. J., Stanley, A. L., Bates, R. C., and Summers, J. E.: Pulmonary arteriovenous fistula. Am. J. Surg., *89:* 1054, 1955.
9. Yater, W. M., Finnegan, J., and Griffin, H. M.: Pulmonary arteriovenous fistula (varix). J. A. M. A., *141:* 581, 1949.

MASSIVE TRAUMA OF THE HIP JOINT*

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The most violent of blows to the hip joint produce injury of a severely destructive nature. This joint is subject to a wide variety of injury from simple fracture of the femoral head, posterior or central dislocations, or acetabular fractures, to combinations of these which may disorder the hip joint seemingly beyond possibility of salvage. That combination which has been found to carry with it a worse prognosis includes combined dislocation with fractures of the femoral head or neck, and occasionally with acetabular damage as well. It is to these that this study has been directed. Since 1953 we have seen 8 cases representing this type of injury.

The text books of 3 or 4 decades ago do not mention such trauma. Its increase parallels that of the automobile, and seems to be associated with the more recent advent of high powered motor cars and excellent highways. All of our cases were the result of auto wrecks, and insofar as the memory of the patients was accurate, or concomitant injuries in the same limb indicated, the causative mechanism was a blow against the knee or thigh which drove the femur backward, where the force was expended in rupture of the bony and capsular structure of the joint. Before 1953 this type of injury, in our hospital, was rare and this probably reflects differences in population and traffic of this period. In 1948 Urist[1] studied 27 posterior fracture-dislocations of the hip and found that 4 of them were of the massively damaged category. Later, in 1954, an extensive survey of 128 injuries of the hip involving dislocations was made by Stewart and Milford,[2] which allowed these investigators to classify with clarity the anatomical varieties of the injury. They found 8 cases of this type and termed these fracture-dislocations "grade IV," by which they are popularly called today.

In evaluating our 8 cases it was desired to learn if particular forms of treatment offered a resonable prognosis or, on the other hand, offered a poor one. Such an assessment is difficult because in so severely injured a patient the presence of other injuries often compromises ideal treatment to one part and it influences the ultimate outcome by inhibiting the use of the limb or prolonging the convalescence. For purposes of discussion, the 8 cases may be considered under three groups (table 1).

Group I. Only 1 patient was encountered in this group, a 20-year-old man with posterior dislocation of the hip and fracture of the inferior non-weight bearing part of the femoral head (fig. 1*A* and *B*). The acetabulum was not damaged visibly. Under general anesthesia, the dislocation was reduced within 2 hours of the accident, the fracture fragments reducing with it without blocking or limiting motion in the joint. He walked on crutches at 6 weeks then resumed full weight bearing at 3 months. The fracture united in good position and 9 months after injury he walked without pain and with no more than 25 per cent limitation of motion. The x-ray showed no significant arthritis so at this time the results were considered good.

Group II. In this group there were 2 patients, aged 47 and 54, whose injuries were similar in every way. In these patients the posterior dislocation of the hip was sustained with fracture of the inferior non-weight bearing part of the femoral head, but in addition the posterior and superior rim of the acetabulum was severely comminuted with resulting instability following reduction. Both of these dislocations were reduced within a few hours of the accident and both patients were immobilized only until soft tissue healing occurred. Thereafter they became ambulatory without weight bearing, one on crutches and the other in a caliper brace, until it was felt that maximal bony healing had occurred. At 14 and 19 months, respectively, they showed upward migration of the acetabulum with moderate arthritic change of the socket and femoral head. Both required canes and walked with moderate pain. There was limitation of joint motion in excess of 50 per cent. In both cases results are considered poor. Of interest is the occurrence in one of these patients of marked

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TABLE 1

Summary of cases

Patient (Age)*	Type of Injury	Treatment	Follow-up Period	End Result
			months	
Group I				
Case 1 (20)	Posterior fracture-dislocation, grade IV, with small fracture of inferior femoral head	Manipulative reduction and plaster abduction boots	9	Good
Group II				
Case 2 (54)	Posterior fracture-dislocation, grade IV, with small fracture of inferior femoral head. Markedly comminuted labrum	Manipulative reduction and plaster abduction boots. Crutches until healed	14	Poor
Case 3 (47)	Posterior fracture-dislocation, grade IV, with small fracture of inferior femoral neck. Markedly comminuted labrum	Manipulative reduction and traction. Caliper brace until healed	19	Poor
Group III				
Case 4 (46)	Posterior fracture-dislocation, grade IV, with large fracture of inferomedial femoral head	Replacement with Thompson prosthesis	8	Fair
Case 5 (17)	Fractures, comminuted, of superior femoral head and posterior rim of acetabulum	Excision of superior fragment of femoral head	13	Fair
Case 6 (49)	Comminuted fracture of femoral head with posterior dislocation, grade IV	Replacement with Thompson prosthesis	9	Fair
Case 7 (65)	Comminuted fracture of femoral head with posterior dislocation, grade IV	Replacement with Thompson prosthesis	24	Fair
Case 8 (53)	Posterior fracture-dislocation with marked comminution of femoral head, neck and trochanter, grade IV	Replacement with McBride prosthesis	10	Fair

* All patients were white men except case 6, a white woman.

FIG. 1*A* and *B*. Group I, case 1. A small fragment fractured from the inferior femoral head; this piece reduced with the reduction of the dislocation and united without functional impairment.

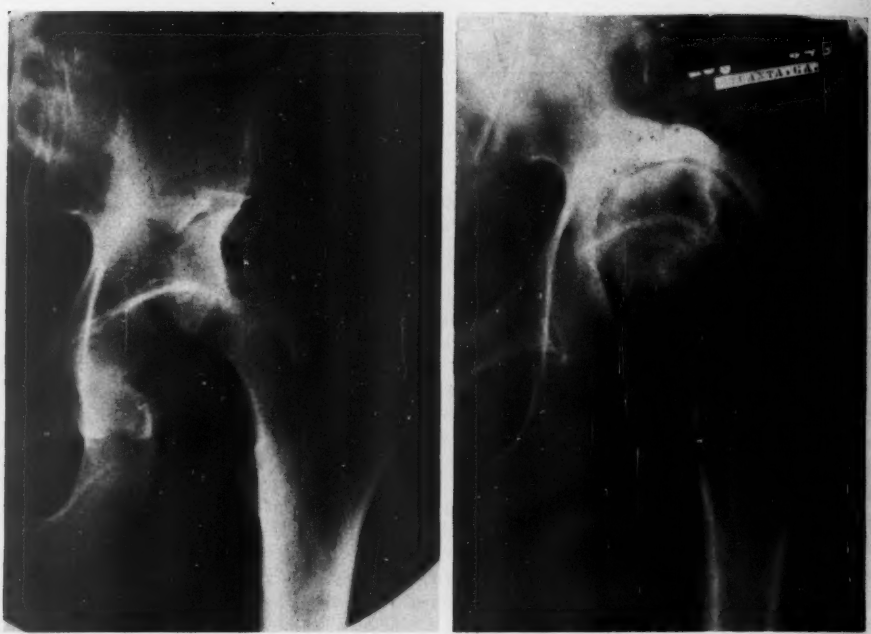

FIG. 2. Group II, case 3. Comminuted fragments of acetabulum not fixed. After 19 months there is upward migration of the acetabulum. Note extracapsular calcification.

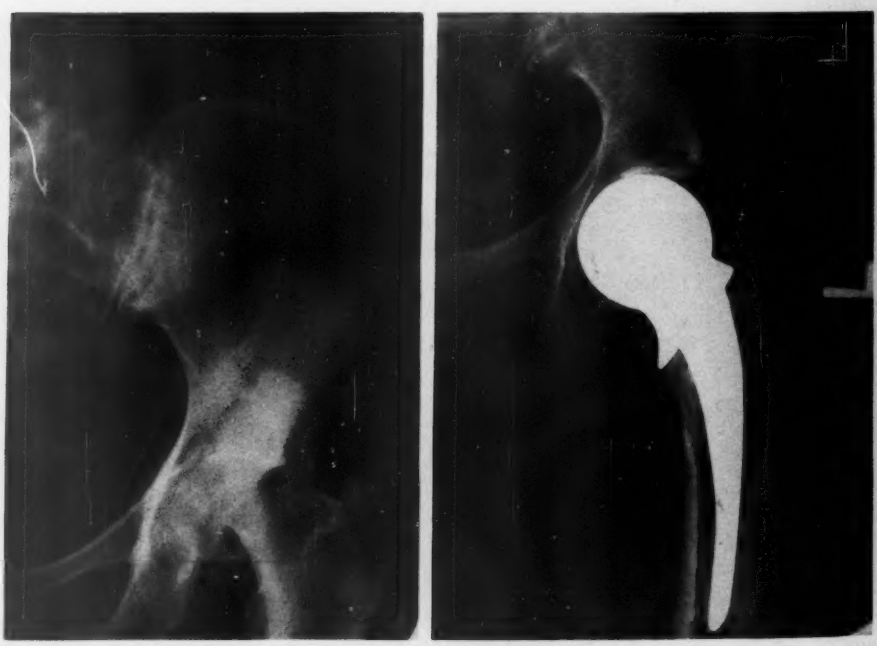

FIG. 3. Group III, case 6. Typical injury of the group that had prosthetic replacements

FIG. 4. Group III, case 8. Severely comminuted femoral head, neck, and trochanter which was repaired with a McBride prosthesis. At 10 months had a good range of motion but moderate pain and an abductor limp.

FIG. 5. Group III, case 5. Result 13 months after excision of fragments from the superior surface of the femoral head. A temporarily functional hip but with developing traumatic arthritis.

periarticular calcification superiorly and posteriorly (fig. 2).

Group III. The remaining 5 patients all had posterior dislocations (except for 1 whose pattern of bony injury was so unusual that it was felt that he should be included with this group) and fracture of the femoral head or neck involving the weight bearing surfaces. Occasional but not clinically significant comminution of the acetabular labrum was seen. The 4 patients who may be strictly classified as grade IV, and whose ages were from 46 to 65 (average of 55), were all treated by prosthetic replacement of the shattered femoral heads (fig. 3). It is notable that the acetabulum was largely spared in those in which the femoral head or neck disintegrated. All of these developed mild to moderate pain upon long periods of weight bearing, and stiffness upon arising in the morning or after sitting. They all had abductor limps and motion was limited to 25 to 50 per cent of normal (fig. 4). Each is rated as a fair result after follow-up periods ranging from 8 to 24 months (average of 12 months).

The other one presented a special problem. Case 5, a 17-year-old boy, had fracture of the superior weight bearing surface of the femoral head with mild comminution of the posterior-superior rim of the acetabulum (fig. 5). It was elected to excise only the fractured fragment of the head and defer more extensive surgery; 13 months after injury he walked with only moderate limitation of motion in flexion and internal rotation, and he has no significant pain. X-ray examination revealed a moderate arthritis developing, but intervention now seems improper.

DISCUSSION

Although certain tentative patterns of treatment emerge from even so small a group, generalization is not justifiable. In cases like group I, it seems reasonable to treat conservatively, barring interference by the fractured fragment of either reduction or motion; this 1 case did well. Those of group II, in which the acetabulum was unstable, appear to merit open reduction with fixation of the acetabular fragments, since the 2 cases in which this was not done did very poorly. This agrees with other studies in which fixation of the acetabular fragments greatly improved the prognosis. Cases 4 to 8 (group III) required either replacement of the femoral head fragments or major reconstructive procedures. In view of the more advanced age in this group, prosthetic replacement was used in each case, and all did fairly which is not greatly different than one finds in fresh femoral neck fractures treated and managed similarly with due allowance for the effect of concomitant injuries. The last was a special case illustrative of the bizarre nature of severe hip trauma and of individualized treatment. So small a series as this certainly does not begin to exhaust the possibilities of treatment, but it is hoped that this report may serve as a stepping stone to the more enlightened handling of these severely injured patients.

Acknowledgment. Thanks is expressed to Dr. Randolph Smith for kind permission to include two of his patients in this study.

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REFERENCES

1. Urist, M. R.: Fracture-dislocation of hip joint; nature of traumatic lesion, treatment, late complications, and end results. J. Bone & Joint Surg., *30-A:* 699, 1948.
2. Stewart, M. J., and Milford, L. W.: Fracture-dislocation of hip; end-result study. J. Bone & Joint Surg., *36-A:* 315, 1954.

MESENTERIC CYSTS*

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Mesenteric cysts are encountered so infrequently that they are rarely considered in differential diagnosis of abdominal masses or of the acute abdomen. The most extensive single experience with mesenteric lymphatic cysts was reported in 1953 by Gross,[4] with 10 cases of isolated cysts. Beahrs and associates[1] reported that only 7 cases of cystic lymphangiomas of the mesentery were recorded in more than 1,000,000 patients at the Mayo Clinic. These unsuspected cysts are usually discovered during exploratory laparotomy performed because of complications arising from them or for unrelated conditions.

Various classifications of true or primary mesenteric cyst have been suggested; however, Ewing's[3] classification is the most simple and complete. In his consideration all true cysts of the mesentery fall into four groups: (1) lymphatic or chylous; (2) enteric; (3) urogenital; (4) dermoid and teratoid. The last two types, urogenital and dermoid or teratoid, are seldom seen but are included for completeness of classification. Gross[4] refers only to the first two types. Pseudocysts of the mesentery may be due to inflammation, hemorrhage or trauma.

Lymphatic, or chylous cysts, are found in any portion of the mesentery or the mesocolon, from the base of the mesentery out to the enteric border. However, most of them are found between the peritoneal leaves of the mesentery of the jejunum and ileum. Lymphatic cysts are always separate from the bowel wall, with a line of cleavage between the two structures. These cysts have an independent blood supply and can usually be excised without injury to the bowel or its blood supply. In size they vary from small swellings of the mesentery to very large tumors which may fill a major portion of the abdominal cavity. They are usually single, multilocular and of a flabby consistency. Their walls are usually quite thin. However, as in the case reported here, the walls may be thickened with fibrous tissue which, on microscopic examination are seen to contain many lymphocytes, lymph follicles and dilated lymph space. These walls consist of connective tissue with no muscular coat or mucosal lining. However, Ewing[3] states that the walls and septa within the lymphatic mesenteric cyst may contain sufficient smooth muscle to make differentiation from enteric cyst on this basis difficult. Several cases of chylous cysts have been reported in which the walls of the cyst were composed of isolated and irregular collections of smooth muscle bundles, but none of these cysts had a true muscular coat, such as seen in congenital duplication of the small intestines.[5, 6] The lining membrane affords the most important distinguishing histologic feature between these two types of mesenteric cysts. In well preserved specimens, a layer of flattened endothelial cells can be identified on the inner surface of a chylous cyst. The lining of recognizable endothelium may be hyperplastic. These cysts usually contain either a clear, serous and watery fluid, chyle, or more inspissated material varying in consistency from milk to thick cream. Gross[4] believed that the more proximal the location of a cyst in the small bowel, the more likely it is to be chylous, because of the higher fat contents of the lymph draining from the jejunum. In his series, the contents of the cysts found in the distal ileum and colon were serous. Sometimes necrosis or old blood are found within the cyst due to embarrassment of the circulation.

Gross[4] also refers to multiple cystic disease of the mesentery in contrast to the isolated mesenteric cysts. In this rare condition, the entire mesentery is filled with myriads of chylous cysts which cannot all be removed. The prognosis is poor in patients with this condition.

The etiology of lymphatic cysts seems to be explained best by the theory proposed by Gross.[4] He considers it likely that they develop from misplaced bits of lymphatic tissue which proliferate and accumulate fluid because they do not

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possess communications which would allow them to drain properly into the remainder of the lymphatic system.

Enteric cysts, or duplications of the bowel, constitute a rather well defined group of mesenteric cysts. They are most frequently seen in the mesentery of the ileum and are usually connected with the intestine or so intimately asso-

FIG. 1. Duplication of small bowel within the leaves of the mesentery, intimately attached to the intestine, and with the same blood supply as the adjacent bowel.

FIG. 2. Cross section of duplication of small bowel with no communication with lumen of alimentary tract.

ciated that the two cannot easily be separated (fig. 1). Since both are served by the same blood supply, removal of the cyst would impair the blood supply of the intestines in that area. The cavity is usually single and the contents are mucinous (fig. 2). The wall is thick and contains a layer of well differentiated smooth muscle and a lining of mucous membrane. Secondary changes in the cyst may destroy much of the epithelial lining.

Urogenital cysts are rare and occur chiefly in women. These cysts are of large size, single or multilocular, involving the mesentery and adjacent regions or extending into the pelvis. They are filled with a brownish, serous fluid containing pseudomucin. The wall is composed of fibrous tissue, and the lining is of cylindrical or cuboidal glandular epithelium. It may contain structures resembling those found in the embryonal kidney. Ewing[3] states that the origin of these cysts is uncertain but that they may be derived from aberrant remnants of the Wolffian body.

Dermoid cysts of the mesentery are also rare. They characteristically reveal some advanced differentiation of one or more types of skin appendage in their wall or contents. They may be located in any portion of the mesentery and their dimensions may be considerable. They are usually lined by squamous epithelium and contain hair and sebaceous material. A few teratoid tumors of the mesentery have been described. Their sources may be ovarian, arising from separation of an original ovarian tumor, or developing from a supernumerary ovary or through implantation from a ruptured ovarian dermoid.

Preoperative diagnosis of a mesenteric cyst is rare.[7] There are no pathognomonic signs or symptoms. The possibility of mesenteric cysts should be considered in the differential diagnosis of all mobile intraabdominal tumors. Symptoms depend on the location and size of the cyst and on the complications which may develop. Most commonly they are produced by pressure from the intraabdominal tumor, by peritoneal irritation following rupture or secondary infection or by intestinal obstruction secondary to complications from the cysts. Mesenteric cysts may fall into 5 groups: (1) those that are discovered during operation for some other condition or are found at autopsy; (2) those that present as a painless enlargement of the abdomen; (3) those that cause pressure symptoms manifested by vague abdominal pain and occasional nausea and vomiting; (4) those that cause intestinal obstruction due to angulation of the gut, adhesions or volvulus; and (5) those that produce symptoms of an acute abdomen following rupture and possible hemorrhage, or secondary infection. Burnett and co-workers[2] reviewed the history in 71 cases and found that pain was the presenting symptom in 82 per cent of the cases. Pain may be caused by pressure, traction on the root of the mesentery, obstruction, rupture, infection and necrosis.

Mesenteric cysts have been found in all age groups. In adults they are usually tense on palpation, whereas in children they may be difficult to feel. A large cyst may be confused with ascites. In the group reported by Burnett and associates,[2] a mass was palpable in 58 per cent of the 71 patients. The size of the cyst, the position and thickness of its wall, and the size of the patient all have a bearing on the palpability of the mass.

X-rays are usually of little value in excluding the possibility of certain other lesions. However, they are useful in determining the position of the mass in relation to neighboring organs if the cyst is large enough to displace the intestines.

The treatment of mesenteric cysts is surgical. At laparotomy it is usually possible to determine grossly the type of cyst. Enucleation is one method of treatment for lymphatic cysts that is often recommended. However, if the mesenteric vessels to the neighboring intestine are damaged, a segment of the bowel may have to be resected. Also, a lymphatic cyst is sometimes so intimately attached to the bowel that resection of the bowel seems to be the simplest procedure. The morbidity and mortality today are low enough following resection that this procedure may be less dangerous than simple enucleation.

Aspiration of a lymphatic cyst as a definitive treatment is to be condemned. Marsupialization has been practically abandoned because a sinus may drain for a prolonged period or it may not close. Nevertheless, marsupialization may be indicated in a rare instance, such as in a poor risk patient. With complications such as volvulus, extensive resection of infarcted bowel may be required, as in the case reported below.

If the cyst is a reduplication of the bowel, resection of both the adjacent bowel and the cyst will be necessary because of the common

blood supply. The type of surgery carried out for other types of mesenteric cysts is dictated by the findings.

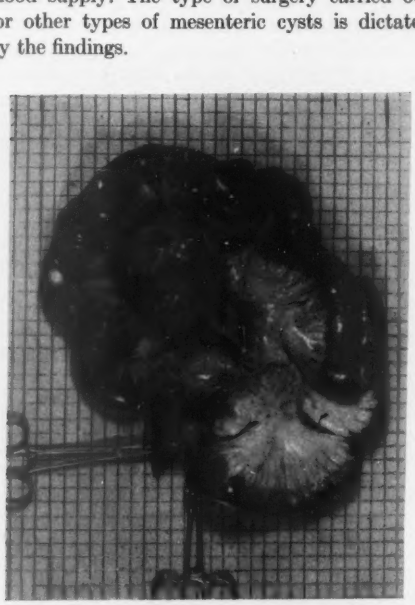

FIG. 3. Lymphatic mesenteric cyst which caused volvulus of small bowel with resulting gangrene.

CASE REPORT

A 47-year-old woman was admitted to Emory University Hospital, March 20, 1957, with the chief complaint of generalized abdominal pain. This symptom had its onset 4 days before admission, in the form of generalized cramplike pains that increased steadily in intensity. Two days before the patient was hospitalized, her abdomen became distended and this also became increasingly severe. The next day nausea developed with several episodes of vomiting. Since the onset of her illness, the patient had had no bowel movements.

A few hours before she was referred to the surgeon her abdominal pains became quite severe, necessitating frequent dosages of narcotics. At the time of surgical consultation, the patient showed symptoms of moderate shock and was gravely ill.

The patient gave a history of similar attacks of abdominal cramping pains during the year before her present illness, but the pain had never been severe. She usually had slight diarrhea associated with these attacks. She had noticed a mass in her lower right abdomen for several months before her present illness. On several occasions before admission, the patient had been hospitalized because of chronic lung disease. Her treat-

FIG. 4. Cross section of multilocular cyst filled with grayish creamlike substance

ment had consisted of numerous bronchoscopies and antibiotics.

Patient's white blood cell count was 19,600 with 86 per cent segmenters. Urinalysis and serum amylase were within normal limits. A roentgenogram of the abdomen revealed moderate distention of several loops of the small bowel, with fluid level. There was a small amount of air within the colon. No free air was seen in the peritoneal cavity. Decompression of the stomach with a Levine tube and suction yielded a large amount of fecal smelling fluid.

For shock, the patient was treated with vasoconstrictors, intravenous fluids and whole blood. In approximately 3 to 4 hours her blood pressure returned from shock level to 120/70. An abdominal exploration was carried out through a right paramedian incision. A large amount of free serous fluid was present in the peritoneal cavity. A large, firm cyst measuring approximately 8 to 10 cm. in diameter was located in the mesentery of the midileum. Further exploration revealed a volvulus of the small intestine, involving the distal jejunum and ileum. Advanced gangrene was present in the lower jejunum and entire ileum. The mesenteric cyst and approximately 60 per cent of the small bowel were resected (fig. 3). An end-to-end anastomosis was performed between the distal jejunum and the ileum, approximately 2 cm. from the cecum. The patient withstood the procedure fairly well. At the end of the operation a tracheotomy was also performed because of the patient's severe, chronic lung disease.

Vasoconstrictors were administered for approximately 36 hours and thereafter her blood pressure remained at approximately 110/60. After peristalsis returned, within 3 or 4 days, the Levine tube was removed, and she was able to tolerate her diet without difficulty. The tracheotomy tube was removed 14 days after the operation. On the 20th day, she was dismissed from the hospital. At this time she was having 3 to 4 loose bowel movements per day.

Pathologic studies of the specimen revealed a roundish shaped tumorlike mass, measuring 8 cm. in diameter, in the mesentery of the terminal ileum. The mass was covered by a thickened wall with areas of infarction. It was rubbery in consistency. On cross section (fig. 4) the mass was found to be a multilocular cyst, with a lining 1 to 1½ cm. thick. There was no communication with the intestinal lumen. The cyst was filled with a thick, grayish creamlike substance. Microscopic examination showed that the wall of the cyst consisted of connective tissue. Necrotic debris was adherent to the inner surface and in a few areas a layer of flattened endothelial cells were identified. No muscular coat was noted in the wall of this cyst. Numerous widely dilated lymphatic channels were seen. There were many areas of infarction throughout the cyst. The specimen also contained approximately 7 feet of gangrenous small bowel, showing a few centimeters of viable intestine at the lines of resection.

In the 2 years since the patient left the hospital, she has tolerated a regular diet and has had from two to three soft stools per day. She has had no nutritional problems.

SUMMARY

The four types of mesenteric cysts: lymphatic or chylous, enteric, urogenital, and dermoid and teratoid are discussed briefly in regard to classification, clinical findings and treatment. Major emphasis is given to the lymphatic or chylous cyst. Since these cysts produce no pathognomonic signs or symptoms, they are usually discovered during exploratory laparotomy performed because of complications arising from them or for unrelated conditions.

In the case reported here, a lymphatic cyst caused volvulus of the small bowel with resulting gangrene, necessitating resection of approximately 60 per cent of the small bowel as well as the cyst.

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REFERENCES

1. Beahrs, O. H., Judd, E. S., Jr., and Dockerty, M. B.: Chylous cysts of the abdomen. S. Clin. North America, *30:* 1081, 1950.
2. Burnett, W. E., Rosemond, G. P., and Bucher, R. M.: Mesenteric cysts. Arch. Surg., *60:* 699, 1950.
3. Ewing, J.: *Neoplastic Disease*, Ed. 4, p. 1062. W. B. Saunders Company, Philadelphia, 1940.
4. Gross, R. E.: *The Surgery of Infancy and Childhood.* W. B. Saunders Company, Philadelphia, 1953.
5. Handelsman, J. C., and Ravitch, M. M.: Chylous cysts of the mesentery in children. Ann. Surg., *140:* 185, 1954.
6. Moore, T. C.: Congenital cysts of the mesentery. Ann. Surg., *145:* 428, 1957.
7. Steinreich, O. S.: The diagnosis of mesenteric cysts. Ann. Surg., *142:* 889, 1955.

A STUDY OF FATAL BREAST CANCER*

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INTRODUCTION

The majority of patients with carcinoma of the breast eventually die of causes related to residual, recurrent or metastatic cancer when followed for greatly extended periods. Numerous studies have been reported concerning various treatment methods which analyze "cures" or survivals following the initial treatment. Collins[2] stated that the results of such studies are remarkably similar despite differences in the three major modes of therapy, *i.e.*, radical mastectomy alone, radical mastectomy plus x-ray, simple mastectomy plus x-ray, and that any greatly different result should be closely scrutinized for an element of patient selection or statistical refinement. Particularly with breast cancer, it is difficult to interpret data relating to cures and survivals, since many patients have asymptomatic rests of viable cancer cells which may lie dormant for long intervals, only to become aggressively active several years later. Other patients may have clinical evidence of disease for several years but carry on with their normal activities. Another reason for difficulty in evaluating statistics on surviving patients is that, although their treatment has failed to eradicate their disease, it has succeeded in making them comfortable. Statistic analysis of a patient's comfort is an extremely difficult task. If one were to follow all breast cancer patients to their death in a current series, it would take many years before the study would be complete, due to the slow insidious nature of this disease in some patients. In order to circumvent some of these difficulties, this problem has been viewed from a different standpoint by studying not the patients who have survived, but rather those who have died. In this study no attempt has been made to compare one form of therapy with another. We are merely trying to see what can be learned about the course of residual, recurrent, or advanced breast cancer by following this select group of patients to their death. From this different viewpoint, it may also be possible to resolve some of the conflicting opinions stated in the literature regarding the effects of treatment.

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MATERIALS AND METHODS

During the period from 1937 to 1957, 1111 patients were seen at the Robert Winship Memorial Clinic with breast carcinoma. Of these patients, 601 had received their initial treatment elsewhere and were referred here for x-ray therapy or for supportive care for advanced recurrent disease, or were sent here for consultation only. Approximately 90 per cent were private patients and 10 per cent were charity patients. All patients were white women. There were 510 patients who were seen for their initial therapy at this clinic; 239 of this group have died. This report will deal only with this group of 239 women. This represents our total experience with fatal breast cancer treated initially at our institution. Those patients treated initially elsewhere were excluded because of inadequate treatment and follow-up data.

Of the 239 patients, 170 met our prevailing criteria for radical mastectomy and were so treated. Our criteria for operability during this 20-year period were not quite so strict as those outlined by Haagensen.[4] Our over-all management regarding operability and operative technique has remained uniform throughout this period. Briefly, all patients were deemed inoperable if they exhibited one or more of the following findings: (1) distant metastases; (2) supraclavicular or fixed axillary metastases; (3) inflammatory carcinoma; (4) extensive local disease with fixation, ulceration, satellite nodules, or extensive edema. Patients with carcinoma during pregnancy or lactation have been managed on the basis of the extent of their disease as outlined above, irrespective of the present or recent pregnancy. It has been our policy to give postoperative radiotherapy to those patients in whom positive axillary nodes were proved microscopically.

TABLE 1

Survival of 258 radical mastectomy patients (Robert Winship Memorial Clinic, 1937–1950)

Years following Radical Mastectomy	Percentage of Patients with No Axillary Node Involvement	Percentage of Patients with Axillary Node Involvement	Over-all Percentage Survival for Both Groups
5	71	46	56
10	44	28	36

Our survival statistics are presented merely for the sake of completeness in case one wishes to compare this study with other groups. These are illustrated in table 1. The operability rate was 82 per cent.

The remaining 69 patients of the 239 dying with breast cancer were judged inoperable and were treated much in the same manner as recurrent disease in the operable group, that is, with x-ray therapy, hormones, extirpative procedures, chemotherapy, supportive care and combinations of the above. Each of the charts of these patients was personally reviewed by the authors and the data were transferred to IBM cards. The results of the various studies on these 239 patients are presented here in tabular and graphic form. Longevity in these studies was computed by the direct or *ad hoc* method as outlined by Berkson and Gage.[1] This method was used, since all patients were followed until their deaths and no patients were lost to study.

RESULTS

The mean age of the over-all group of 239 patients was 53 years. The mean age of the 170 patients receiving radical mastectomy was 51.8 years. The mean age of the 69 cases treated by methods other than radical mastectomy was 57 years.

The graph in figure 1 depicts the longevity of the operable patients from their initial treatment to death. Those with no involvement of the axillary nodes appear to have a better outlook in terms of longevity. However, due to the small number of node negative patients and the atypical slope of their longevity curve, this group was subjected to further analysis. The value of χ^2 was calculated to be 1.12 at the point of maximal divergence of the two curves. The probability that this difference in longevity is due to chance, and not due to the stage of their disease, is approximately 30 per cent. Therefore, in this particular graph even the greatest difference in longevity between the node negative and node positive group is not statistically significant. We have also plotted the expected death rate[5] for a group of white women of the same age dying from all causes during the same period of time. In this graph, as well as in figures 2 and 3, the 18 patients dying from causes other than breast cancer have been deleted. Of these, 9 died before 5 years and 9 died after 5 years following mastectomy. Postmortem studies in a few of these revealed no evidence of recurrent carcinoma. The other patients may or may not have had recurrent cancer and we cannot tell whether the "pneumonia," "cirrhosis," or "stroke" on their death certificates was actually due to metastatic disease. Four of the patients died of leukemia; 3 of these received previous x-ray therapy. It would be outside the scope of this report to discuss this relationship between radiation and leukemia.

In figure 2 all patients have again been followed to their deaths. We have computed longevity of the inoperable group, from the initial treatment to death, with the surgically treated group. In the radical mastectomy group longevity was computed from the initial recurrence of cancer to death. These two groups then become similar in certain respects. They often present with the same clinical picture, such as multiple bony or pulmonary metastases, or generalized carcinomatosis. They also received similar therapeutic regimes, such as x-ray therapy, and so forth. Treatment in both groups was primarily for palliation. When one considers the different mean ages in the operable and inoperable groups in figure 2 the slight difference in longevity as shown becomes more pronounced. From these apparently paradoxical curves two or more conclusions might be drawn, as shown in figure 2:

1. When recurrent breast cancer becomes clinically apparent following radical mastectomy, it is more aggressive when measured in terms of longevity. This seems valid since this group has had cancer for several months or years and many have disseminated disease at the time the recurrence becomes clinically apparent (table 2).

2. One might conclude that radical surgery has a deleterious effect on the patient, since the group treated without surgery has a higher

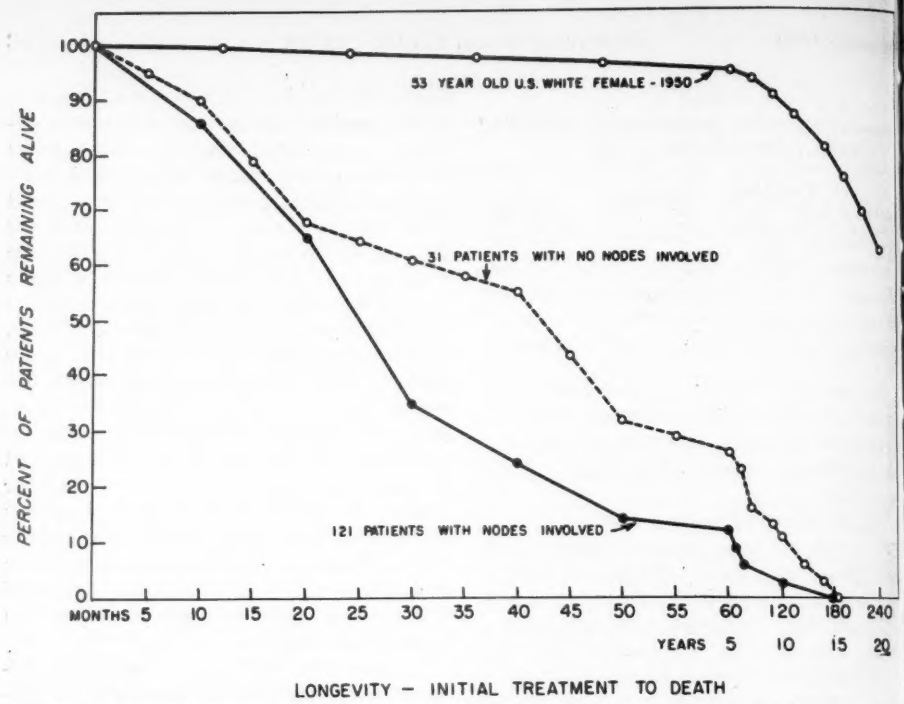

FIG. 1. Influence of stage of disease on longevity in 152 radical mastectomy patients dying with breast cancer.

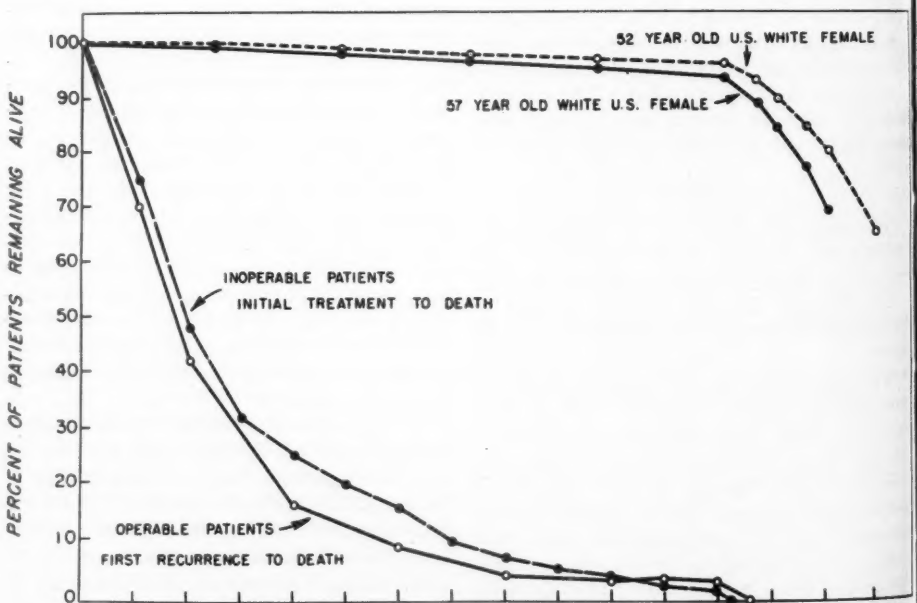

FIG. 2. Longevity of operable patients following initial recurrence, and inoperable patients following initial treatment.

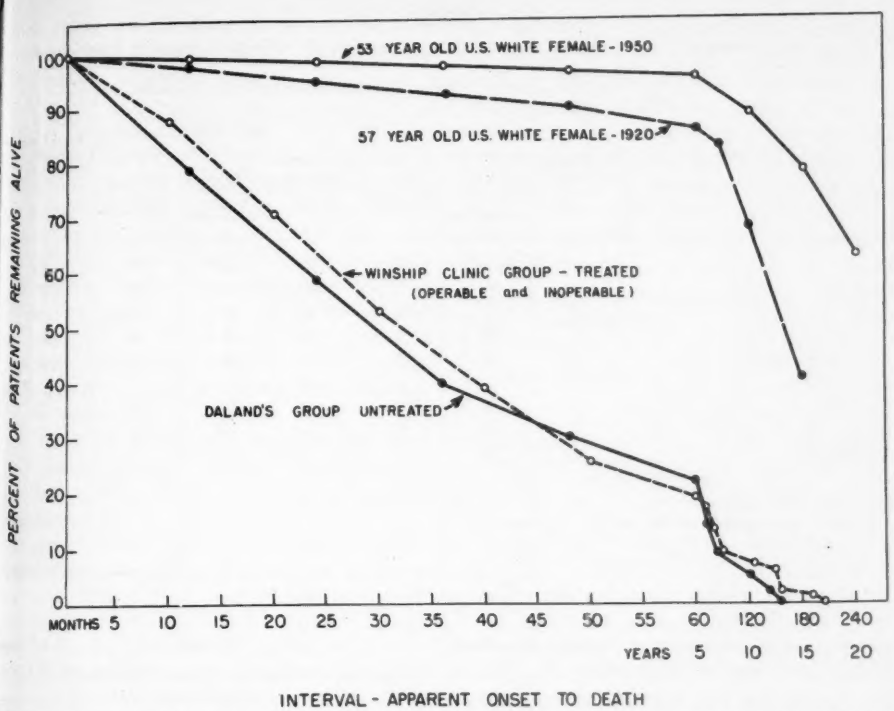

FIG. 3. Longevity of Daland's 100 untreated cases, and 221 treated patients (operable and inoperable) dying with breast cancer.

longevity curve. Let us bear in mind, however, that the surgically treated patients have a variable period of time following radical mastectomy, in which they are symptomatically and clinically free of disease. In those patients dying with breast cancer the mean period free of disease following surgery was 23 months.

The graph in figure 3 compares Daland's[3] series of 100 untreated cases dying of breast cancer with our over-all group (operable and inoperable patients) of treated patients dying with breast cancer. These two groups are similar in certain respects; all patients died in both groups. Other than the fact that they all died with breast cancer, they are both unselected groups with all stages of operable and inoperable disease. Longevity for both groups is computed from the apparent onset of disease (that is, when the patient first noticed the breast lump or other initial symptom) to death. From these longevity curves there appears to be little or no significant effect from the treatment. When one considers that Daland's cases were slightly older than ours and that these were reported over 30 years ago when the longevity in the normal population was not so good as now;[6] indeed, the untreated patients might appear to live even longer! Again, we must remember that no account has been made of the patients' comfort or of the symptom free period, which averaged 23 months in our surgically treated patients. As Wade[7] has stated, "If the clinical pictures of the untreated and treated groups are compared, some of the treated group were symptom free or disease free for at least part of their survival period (of 38½ months). We may, therefore, describe them as 'enjoying' a survival period of so many months (or proportion of it), whereas the untreated group might be more accurately said to 'endure' a survival period of 38½ months."

Table 2 summarizes the data pertaining to the initial recurrence of cancer in our 170 patients dying following radical mastectomy. In 19 per cent of the node negative group the first

TABLE 2

Longevity according to site of initial recurrence or metastasis in patients dying with breast cancer

Site of First Recurrence	Carcinoma Confined to Breast, Stage I			Metastasis to Axillary Nodes, Stage II			Total Number of Patients in Stages I and II
	Number of patients in group	Mean interval in months		Number of patients in group	Mean interval in months		
		Initial treatment to first recurrence	First recurrence to death		Initial treatment to first recurrence	First recurrence to death	
Local	4	10	10	22	15	12	26
Regional	3	16	3	24	22	24	27
Bony	10	30	16	29	20	11	39
Generalized	4	88	11	13	20	7	17
Lung and pleura	5	44	12	15	35	8	20
Mediastinum	0			2	15	7	2
Liver	2	8	3	15	14	3	17
Brain	2	36	5	1	16	4	3
Abdominal	1	18	61	0			1
Died of other causes	5	71*		13	50*		18
Totals	36			134			170

* The mean interval in months from initial treatment to death.

recurrence was either local or regional. However, in the group with axillary nodes involved, 35 per cent of the first recurrences were local or regional. This increase in local and regional recurrences over the node negative group is in agreement with other studies. In the 99 patients with distant metastases as their initial clinical evidence of recurrence it is assumed that their first recurrences were probably due to hematogenous spread. The extremely poor prognosis following the detection of either liver or brain metastases is noteworthy. The one patient with abdominal metastasis presented with intestinal obstruction. At laparotomy, metastatic breast cancer was found to be the cause. Her long survival following this diagnosis is interesting. Another point of interest in table 2 is that once cancer recurs the outlook is no better for the node negative group than those who originally had nodes involved; however, the over-all longevity from initial therapy to death might be slightly better in the node negative group, as previously pointed out in figure 1.

Table 3 compares the interval free of disease following surgery with the mean longevity following the first recurrence to death. We were somewhat surprised that the longevity following the first recurrence is not significantly greater even when patients had very long symptom free periods following radical mastectomy. Again, this emphasizes that once disease recurs, longevity following the first recurrence is remarkably constant.

TABLE 3

Longevity from first recurrence correlated with length of "free interval"

Interval in Months Free of Disease	Number of Patients in Groups	Mean Longevity in Months from First Recurrence to Death
0–10	54	11
11–20	36	14
21–30	26	8.2
31–40	9	8.6
41–50	8	12
51–60	3	14
61–70	3	29
71–80	2	12
81 and over	8	18

These findings are sometimes not in agreement with clinical impressions. Two factors probably explain this disparity. First, these fatal breast cancer patients generally have the more biologically active tumors and, therefore, are more resistant to treatment. Secondly, the exclusion

of all living patients also excludes our long term survivors following successful treatment for recurrent or metastatic disease.

SUMMARY

A group of 239 patients dying with breast cancer of varying stages was studied. This represents our total experience with fatal breast cancer treated initially at our clinic from 1937 to 1957. It should be reiterated that this study differs from most others, since only patients dying with breast cancer have been studied, and no living patients have been included. From this relatively small group of patients with operable or inoperable fatal breast cancer, three findings are of special interest:

1. Longevity in this select group, computed from initial symptom or from original therapy to death is not greatly altered by any present treatment.

2. Once breast cancer becomes clinically manifest as a recurrence or metastasis, longevity computed from date of recurrence to death is not greatly affected by the original stage of the disease or by the "disease free" interval.

3. In 99 of the operable patients their initial recurrences were first clinically detected as distant metastases. It is in this group that we expect to increase longevity when methods become available to prevent or to treat blood borne metastases.

It should be re-emphasized that no attempt has been made in this study to evaluate the patient's comfort. Since treatment apparently does not alter longevity in this group of patients, their comfort then becomes the most important goal of therapy. These findings are not to be construed as a condemnation of present therapeutic regimes. Instead, we consider them as a stimulus to further research in the therapy of breast cancer.

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REFERENCES

1. Berkson, J., and Gage, R. P.: Calculation of survival rates for cancer. Proc. Staff Meet. Mayo Clin., *25:* 270, 1950.
2. Collins, V. P.: Reasons for failure among 957 cases of cancer of the breast. In *Third National Cancer Conference Proceedings*, pp. 171. J. B. Lippincott Company, Philadelphia, 1957.
3. Daland, E. M.: Untreated cancer of the breast. Surg. Gynec. & Obst., *44:* 264, 1927.
4. Haagensen, C. D.: *Diseases of the Breast*, p. 538. W. B. Saunders Company, Philadelphia, 1956.
5. Statistical Abstracts of the United States: 1954 (75th edition), Washington, D. C., 1954.
6. Statistical Abstracts of the United States: 1950 (71st edition), Washington, D. C., 1950.
7. Wade, P.: Untreated carcinoma of the breast. Brit. J. Radiol., *19:* 272, 1946.

PARATHYROID CYSTS: REPORT OF A CASE*

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Cysts of the parathyroid gland of clinical significance are extremely rare, only 25 cases being recorded in American and European literature. The condition has been seen more frequently in women, and in reported cases at ages ranging from 16 to 79 years. It occurs with equal frequency on both sides of the neck, and is almost always closely associated with the inferior pole of the thyroid gland. The presenting symptom is usually that of a mass in the neck, although occasionally the size may be sufficient to result in some difficulty in swallowing or respiration. Recurrent laryngeal nerve paralysis due to pressure has been reported. The cysts are not associated with dysfunction of the parathyroid.

Accurate preoperative diagnosis cannot be made, but gross characteristics at surgery are sufficiently distinctive to lead to strong suspicion of the correct diagnosis. A cyst of the parathyroid may be suspected when a cystic lesion is found near the inferior pole of the thyroid gland. It is usually enucleated quite easily, with a distinct plane of cleavage between it and the thyroid. The wall is fibrous but usually thin and translucent. When the cyst is opened it contains opalescent or watery fluid, and the cyst wall is seen to be smooth.

Microscopically, the cysts are distinguished by a wall of fibrous tissue composed of parallel collagenous bundles with relatively few nuclei. The cyst is usually lined with cuboidal or low columnar epithelium, although frequently a distinct epithelial layer is not found. Lymphoid tissue in the wall is occasionally seen, and Nylander[11] reported the presence of muscle tissue in the wall in his case. The diagnostic characteristic, however, is the presence of nests of parathyroid cells in the wall of the cyst.

The clinical and pathologic characteristics of the cysts in reported cases are recorded in table 1.

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CASE REPORT

L. G., a 44-year-old white woman, had been in excellent health, but consulted a physician in July 1957 for a periodic examination. She was found to have a mass in the left lower anterior portion of the neck which moved with swallowing. This was about 3 cm. in diameter and was described as quite firm in consistency. No other physical abnormalities were noted, and urinalysis and routine blood counts were normal. A protein bound iodine determination was 6.5 mg. per 100 gm.

A diagnosis of solitary nodule of the thyroid gland was made, and surgical removal advised. The patient entered Emory University Hospital on August 9, 1957, and repeat physical examination confirmed the previous findings. Operation was done on August 10, 1957.

At the operation, she was found to have a 3 by 2 by 2 cm. cystic mass somewhat posterior and lateral to the inferior pole of the thyroid gland on the left side. The mass was loosely attached to the thyroid and was easily dissected and removed intact. The recurrent laryngeal nerve was posterior and intimately associated with the cyst, but was easily preserved. In addition, the patient was found to have a 1-cm. nodule in the left lower pole of the thyroid and a left hemithyroidectomy was done. No parathyroid gland was found on the left side after diligent search, but a nodule of yellow-orange tissue in the wall of the cyst was thought to be parathyroid. The operative diagnosis was parathyroid cyst and nodular hyperplasia of the thyroid.

The patient's convalescence was uninterrupted and she had no indication of parathyroid or thyroid deficiency. She was dismissed from the hospital on August 15, 1957, and has remained well.

Examination of the surgical specimen revealed a 3 by 2 by 2 cm. cyst with a thin, translucent wall. On opening the cyst it was found to contain thin, opalescent fluid. There were no intraluminal projections, but there was a palpable thickening in one area, measuring 6 mm. in diameter, which had a yellow-orange appearance.

Microscopic study of the cyst (figs. 1 and 2) revealed a fibrous wall composed of parallel bundles of collagenous tissue which was relatively

TABLE 1

Summary of case reports of parathyroid cysts

Case No.	Author	Year	Age	Sex	Side	Position in Relation to Thyroid	Size	Symptoms	Fluid	Lining Epithelium	Parathyroid Cells in Wall	Diagnosis
1	Goris[6]	1905	22	M	L			Mass in neck	Three adjacent cysts containing degenerated parathyroid cells			
2	Anzilotti[1]	1909	25	M	L	Inf. pole	Egg	Mass in neck		Columnar	Yes	Parathyroid cyst
3	de Quervain[4]	1925	55	F	L	Inf. pole substernal					Yes	Parathyroid cyst
4	Nylander[11]	1929	16	M	R	At angle of mandible	Goose egg	Mass in neck	Contained cholesterol and cell debris	Low columnar cuboidal	Yes	Parathyroid cyst
5	McKnight[10]	1946	17	M	L	Inf. pole	Golf ball	Mass, dyspnea, hoarseness	Milky	Scant, flat, endothelial-like	Yes	Parathyroid cyst
6	*Welti[12]	1946	36	F	L	Inf. pole	Orange	Mass, dyspnea, dysphagia, paralysis left vocal cord	Opalescent	Cylindrical	Yes	Parathyroid cyst
7		1946	28	F	L	Inf. pole	Nut	Mass, weakness, bone decalcification	Serous	Cuboidal	Yes	Parathyroid cyst
8		1946	53	F	L	Inf. pole		Mass, weakness left vocal cord		None	Yes	Parathyroid cyst
9		1946	35	F	L	Inf. pole	Mandarin	Mass in neck	Serous	None	No	Parathyroid cyst
10	† Black and Watts[2]	1949	32	F	L	Inf. pole	4 cm.	Mass in neck		Cuboidal	Yes	Parathyroid cyst
11		1949	30	M	R	Sup. pole	1 cm.	Had hyperparathyroidism with adenoma cyst (found incidentally)		Cuboidal	Yes	Parathyroid cyst
12	McGoon and Cooley[9]	1951	66	M	R	Inf. pole	4 x 2 cm.	Found incidentally at surgery for goiter		Cuboidal	Yes	Parathyroid cyst
13		1951	49	F	R	Inf. pole	5 x 4 cm.	Mass in neck	Clear	None	Yes	Parathyroid cyst
14		1951	43	F	L	Inf. pole	4 x 3 cm.	Mass in neck		Cuboidal	Yes	Parathyroid cyst
15	Greene and associates[7]	1952	38	F	R	Right side of neck	9 x 5 x 4 cm.	Mass in neck	Blood tinged	Parathyroid cells	Yes	Cystic degeneration of adenoma
16	Maxwell and associates[8]	1952	39	F	L	Inf. pole	7 x 3 cm.	Mass in neck		Cuboidal	Yes	Parathyroid cyst
17		1952	33	F	R	Inf. pole	9 x 4 x 3 cm.	Mass in neck	Clear	Cuboidal	No	Parathyroid cyst
18		1952	29	F	R	Inf. pole	1.2 x 1 x 0.9 cm.	Mass in neck		Cuboidal	Yes	Parathyroid cyst
19	Crile and Perryman[3]	1953	34	F	L	Inf. pole	2.5 cm.	Mass in neck	Clear	Cuboidal	Yes	Parathyroid cyst
20		1953	56	M	L	Inf. pole	7.0 cm.	Mass, dyspnea		Cells of parathyroid type		Transitional cell cyst of parathyroid
21		1953	69	F	R	Inf. pole and superior mediastinum	6 x 3 cm.	Mass, dyspnea, paralysis vocal cord		None	Yes	Parathyroid cyst

TABLE 1—*Continued*

Case No.	Author	Year	Age	Sex	Side	Position in Relation to Thyroid	Size	Symptoms	Fluid	Lining Epithelium	Parathyroid Cells in Wall	Diagnosis
22	Crile and Perryman[3]	1953	32	F	L	Inf. pole	4 x 3 cm.	Mass in neck	Clear	None	Yes	Parathyroid cyst
23		1953	79	F	L	Inf. pole	6 x 1.5 cm.	Mass, dyspnea		Cuboidal	Yes	Parathyroid cyst
24	Fisher and Gruhn[5]	1957	32	F	L	Inf. pole	2.5 x 2 x 1 cm.	Mass in neck	Opalescent	Flat, cuboidal	Yes	Parathyroid cyst
25		1957	60	M	L	Inf. pole	4 cm.	Found incidentally at autopsy. Had coincidental goiter	Yellow, watery	Flat, cuboidal	Yes	Cyst arising in adenoma
26	Perdue and Martin	1959	44	F	L	Inf. pole	3 x 2 x 2 cm.	Mass in neck	Opalescent	None	Yes	Parathyroid cyst

* Mentions another case, but not described.
† Mentions 4 other cases identical in all respects with the exception that parathyroid tissue was not found in the wall.

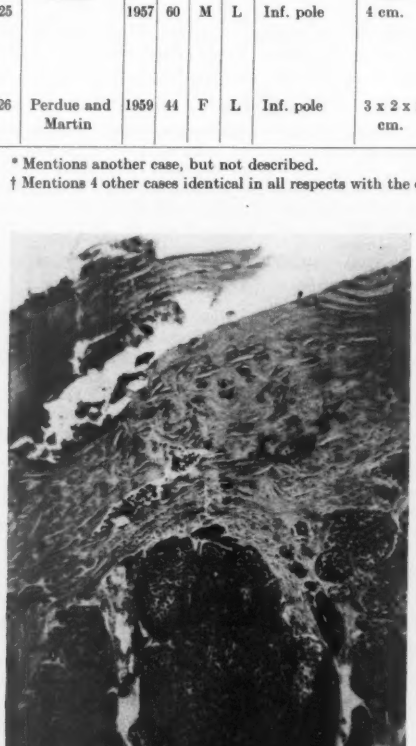

FIG. 1. Photomicrograph, low power, cyst showing fibrous wall, nests of parathyroid cells, and area of metaplastic bone formation.

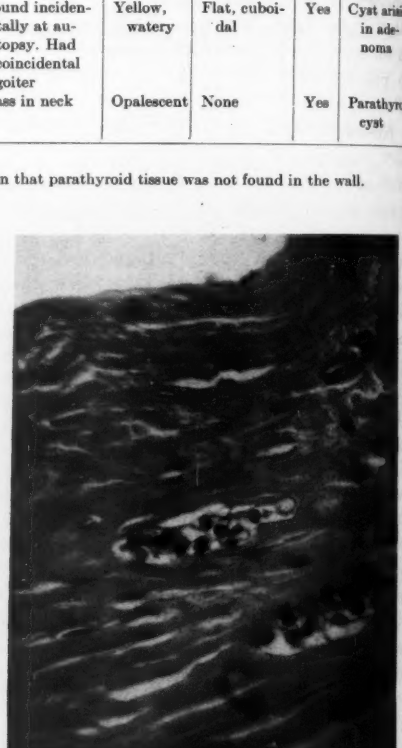

FIG. 2. Photomicrograph, high power, nests of parathyroid cells in fibrous cyst wall.

acellular. There was no identifiable lining layer. In one area of the cyst wall there was a focus of metaplastic bone formation, and nests of parathyroid cells were seen in the wall, with gradual transition to a nodule of typical parathyroid cells. Gross and microscopic descriptions of the thyroid tissue were those of nodular hyperplasia. Final diagnosis was parathyroid cyst.

DISCUSSION

The principal importance of these lesions is that they are space occupying tumors which may grow large enough to give rise to diagnostic question and to the symptoms resulting from pressure on adjacent structures. Difficulty in swallowing and breathing was noted in several of the

cases reported and recurrent laryngeal nerve palsy in 4. Several of the tumors reported extended into the superior mediastinum. They are apparently not associated with disturbed function of the parathyroid, although the patient of Greene and associates[7] did exhibit transient postoperative tetany. In this instance the cyst was apparently cystic degeneration of a benign adenoma, in contrast to the usual case report which gives no indication of the presence of adenoma. It should be noted also that one of Welti's patients[12] had had weakness and bone decalcification for many years before removal of the cyst. The postoperative condition is not given.

There is much discussion in the literature as to the possible etiology of this condition. Several earlier authors, including McGoon and Cooley,[9] Welti[12] and Nylander,[11] believed that these are the end result of expansion of vestigial remnants of the third and fourth branchial clefts which secondarily incorporated adjacent parathyroid tissue in the wall. Nylander's case in particular showed cholesterol and cellular debris in the cyst fluid, and muscle tissue as well as normal appearing parathyroid in the wall. Other reports do not include similar findings. The lesion in this instance was also atypically located at the angle of the mandible.

The preponderance of recent opinion is that these cysts arise from parathyroid parenchyma. Some are apparently the result of cystic degeneration of adenomas, such as one case of Fisher and Gruhn[5] and the case of Greene and associates.[7] Also, Goris'[6] patient, the first reported, had a trilocular cyst containing degenerated parathyroid tissue. It is possible that the majority of such cysts arise by degeneration, but it seems equally possible that retention cysts, which are frequently observed microscopically, may occasionally enlarge sufficiently to be of clinical importance. Transitional stages have not been described, however.

SUMMARY

Parathyroid cyst is a rare lesion which frequently can be recognized at operation by a typical location in the region of the inferior parathyroid, and by the gross appearance of a translucent cyst loosely adherent to the inferior pole of the thyroid.

Microscopically it is characterized by a thin wall composed of parallel collagenous bundles with nests of parathyroid cells identifiable in the wall. It is frequently lined by cuboidal epithelium, although an identifiable lining layer is often absent. The fluid is watery or opalescent in most instances.

Such cysts probably arise from parathyroid parenchyma, either by retention or cystic degeneration. Occasionally a cyst arising from vestigial remnants of the branchial clefts may incorporate parathyroid tissue in the wall.

These are space occupying lesions of the neck, which give rise to compression symptoms and diagnostic concern. They are rarely, if ever, of functional importance.

A representative case history is reported, bringing the total number of recorded cases to 26.

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REFERENCES

1. Anzilotti, G.: Contributo alla patogenesi delle cisti parabranchiali del collo; ciste del ductus parathyroideus. Clin. chir., *17:* 610, 1909.
2. Black, B. M., and Watts, C. F.: Cysts of parathyroid origin: report of two cases and study of incidence and pathogenesis of cysts in parathyroid glands. Surgery, *25:* 941, 1949.
3. Crile, G., Jr., and Perryman, R. G.: Parathyroid cysts; reports of five cases. Surgery, 34: 151, 1953.
4. de Quervain, F.: Chirurgische demonstrationen. (Epithel-korperchencyste). Schweiz. med. Wchnschr., *6:* 1168, 1925.
5. Fisher, E. R., and Gruhn, J.: Parathyroid cysts. Cancer, *10:* 57, 1957.
6. Goris: Extirpation de trois lobules parathyröidiens Kystiques. J. de chir. et Ann. Soc. Belge de chir., v. 394, 1905.
7. Greene, E. I., Greene, J. M., and Busch, R. C.: Unusual manifestations after removal of parathyroid cyst. J. A. M. A., *150:* 853, 1952.
8. Maxwell, D. B., Horn, R. C., Jr., and Rhoads, J. E.: Cysts of the parathyroid; report of three cases clinically simulating nodular goiter. A. M. A. Arch. Surg., *64:* 208, 1952.
9. McGoon, D. C., and Cooley, D. A.: Parathyroid cyst: an unusual cervical tumor; report of three cases. Surgery, *30:* 725, 1951.
10. McKnight, R. B.: Parathyroid cyst. South. Surgeon, *12:* 179, 1946.
11. Nylander, P. E. A.: Über parathyreoideale Halszysten. Acta chir. scandinav., *64:* 539, 1929.
12. Welti, H.: Apropos des kystes parathyröidiens, Mém. acad. chir., *72:* 33, 1946.

RECURRENT CARCINOMA OF THE COLON*

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The possibility of recurrence of carcinoma of the colon following resection is always of concern. Even with all of the available diagnostic aids, it is difficult to recognize its existence. When postoperative obstruction occurs, the logical cause is recurrent disease. However, the early radiographic appearance may present difficulty in distinguishing from recurrent carcinoma and the normal process of healing, because of the unavoidable narrowing of the lumen at the anastomotic site from edema and mucosal deformity. Continuity of the bowel cannot be re-established without some deformity, which has been shown radiographically to last 6 months, or even permanently.

In a recent report, Goldenberg and Hayes[6] stated that postobstruction intestinal dyskinesia may occur and be a cause for concern in the differentiation of recurrent disease. Therefore, constant vigilance should be maintained and proper evaluation be made in order to detect early evidences of recurrence.

In the many reports there is a great variation in the frequency of recurrences, but there are reasons for these discrepancies. The stage of the disease is, or course, of great significance; the presence of metastases, local and distant, has influence; and the invasion of the lymphatics and blood vessels must be taken into consideration. Microscopic examination, both proximally and distally, is vital in determining the extent of infiltration. The type of procedure would limit to some degree the likelihood of invasion, as well as the measures used to control spread of the cancer at operation. Goligher and associates[7] noted a 50 per cent recurrence rate, with two-thirds of the lesions at the suture line. Beal and Cornell[1] reviewed a series of 193 patients with resected carcinomas of the colon and found that 15 per cent had recurrence at the anastomotic site. McGrew and associates[10] reported a 16 per cent local recurrence in a series of 55 cases treated by resection and anastomosis. Cole has noted a 10 per cent suture line recurrence of low sigmoid and rectosigmoid carcinoma following anterior resection and anastomosis.

Gilchrist and David[5] reported a 20 per cent incidence of recurrence, which is considered average. Judd and Bellegie[8] reported 282 cases of recurrence, in which a 5-year follow-up revealed 60 cases with lesions 10 cm. of the dentate line with a recurrence rate of 41.7 per cent; 159 patients had lesions between 11 and 15 cm. of the dentate line, with a 30.2 per cent recurrence rate; and 61 had lesions between 16 and 20 cm. of the dentate line, with a recurrence rate of 16.4 per cent. This is striking evidence of the decreasing incidence of recurrence from the dentate line to the proximal part of the intestine.

In 1950 Garlock and Ginzburg[4] reported a 35.7 per cent local recurrence rate following low anterior resection and anastomosis. Dunning and associates,[3] using the low anterior resection, noted a 22.5 per cent recurrence rate in a series of 102 cases. Judd and Bellegie[8] had an average recurrence rate of 32.8 per cent.

Goligher and associates[7] reported 152 patients treated with anterior resection for carcinoma of the rectosigmoid and noted that 10 per cent had suture line recurrence. However, of 2000 resections for carcinoma of the rectosigmoid, where the rectum and anus were removed, they noted only 3 local recurrences. This difference seems to be of real significance.

Lofgren and associates[9] recently reported 108 cases of locally recurrent carcinoma of the rectosigmoid which showed a 16 per cent higher rate when microscopic sections of the originally removed tumor had revealed lymphatic invasion. It would seem, therefore, that reporting of longevity statistics in the future should include, as previously mentioned, whether lymphatic or blood vessel invasion was involved at the time of resection.

It is evident that the recurrence rate in carcinoma of the colon is alarmingly high. In nearly all cases, evidence strongly suggests that tumor

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cells were left at the time of original operation. Lofgren and associates,[9] in a series of 108 cases, attributed 3 to incomplete resection of the primary tumor, whereas 12 recurrences were thought to be due to incomplete resection of satellite invaded blood vessels and lymphatics. Of 108 recurrences, 76 were thought to be due to local implantation at the time of surgery, which cannot be definitely proved.

The inherent difficulty in preventing local implantation was emphasized by Pomeranz and Garlock.[11] In 20 cases, carcinoma cells were found in 10 per cent of the patients on the serosal surface of the bowel.

In experimental work with rabbits, by Vink,[12] a 44 per cent incidence of carcinoma cell implantation was noted where there had been the usual bowel preparation with antibiotics. In animals which had not had colon preparation with antibiotics, it was noted that 13 per cent had recurrence.

McGrew and associates[10] consistently recovered malignant cells from the lumen of the bowel close to the tumor. Even applying ligatures proximal and distal to the tumor, and allowing ample margin, yielded as high as 8 per cent positive cell recoveries.

Many methods have been advised for prevention of recurrence, or lessening of the likelihood of tumor cells being left at operation. The use of ligatures, proximal and distal to the tumor, before excision is undertaken, has been advised by McGrew and associates.[10] The use of a protective cover over the colon has been proposed by Walt and Waugh[13] and Pomeranz and Garlock.[11] The cover is applied in such a way as to attempt to keep the cells from being squeezed through the bowel wall during manipulation. The desire is to prevent the cells from flowing freely in the abdominal cavity and thus result in implantation.

Walt and Waugh,[13] as well as Goligher and associates,[7] have advised ligation of the inferior mesenteric artery and vein and the accompanying lymphatics before surgical attack on the bowel wall. The belief is that cancer cells already invading veins, lymphatics and arteries of the bowel close to the tumor can easily be broken off and freed into the blood or lymphatic stream during handling of the tumor and its immediately adjacent colon. If these structures are ligated before handling the tumor, spread through the inferior mesenteric artery, vein and accompanying lymphatics in a lesion of the left colon is prevented.

Walt and Waugh[13] have advised the use of mercuric chloride lavage of the distal bowel lumen before performing the anastomosis. However, the harmful effect of the lavage may be greater than the threat of spreading cancer cells and the inability to destroy them.

Resection of the proper amount of bowel, proximal and distal to the tumor, offers greatest protection against future difficulty. Lofgren and associates[9] advised excising a margin of 6 cm. both proximal and distal to the tumor. It is believed that excision of more of the bowel distal to the lesion does nothing to improve the recurrence rate. On the other hand, McGrew and coworkers[10] feel that a 3 cm. margin of bowel excision, both proximal and distal from the lesion can be considered adequate for most cases. The distal segment cannot always be widely resected. A factor such as limited available exposure on low rectosigmoid lesions is a major technical problem.

In the past, elderly patients have not been considered able to tolerate extensive curative surgical procedures. Recent opinion is that these patients will withstand a more radical resection of most malignant tumors, provided the procedure is not too prolonged. This does not suggest that a limited procedure should be advocated with these older patients, but that the necessary procedure should be carried out with dispatch and decisiveness.

It has been shown that carcinoma cells in many patients invade the lymphatics and blood vessels, both locally and systemically. This has caused recent therapeutic efforts to be directed toward destroying such cells in the blood stream as well as in the local area. The limiting factor is the inability to obtain a satisfactory anticarcinogenic agent which is not harmful to the host. The prophylactic use of the anticarcinogenic drugs, such as nitrogen mustard, 2 per cent Chlorpactin and others, has proved of interest experimentally; however, the best method to prevent spread of the disease is adequate resection of the tumor, the adjacent intestine and the regional nodes.

The cases presented demonstrate problems which can be expected following treatment of these conditions.

CASE REPORTS

Case 1. W. P. was admitted to the Atlanta Veterans Administration Hospital in May 1957 because of swelling of the lower extremities of 2 years' duration, for which no cause was found.

On diagnostic study, because of vague associated abdominal discomfort, a barium enema demonstrated a filling defect at the hepatic flexure. This appeared as a pedunculated mass extending into the lumen of the colon. The mass measured approximately 3 cm. in diameter and no additional polyps were seen.

The patient was operated upon and an adenocarcinoma of the hepatic flexure, 3 to 5 cm. in diameter, sessile, with serosal deformity was found (fig. 1). An additional polyp was felt within the lumen of the right colon and a right hemicolectomy and ileotransversostomy were done. On inspection of the gross specimen, two polyps proximal to the large lesion of the hepatic flexure were noted. There was approximately 7 cm. of normal appearing colon distal to the hepatic flexure lesion. No additional polyps were felt within the left colon.

The patient had an uneventful course for the first 6 weeks postoperatively and then passed bright blood from the rectum. All examinations including sigmoidoscopy were negative. Barium enema was repeated twice and found to be negative. Because of persistence of the bleeding, requiring multiple transfusions, the patient was prepared and reoperated upon.

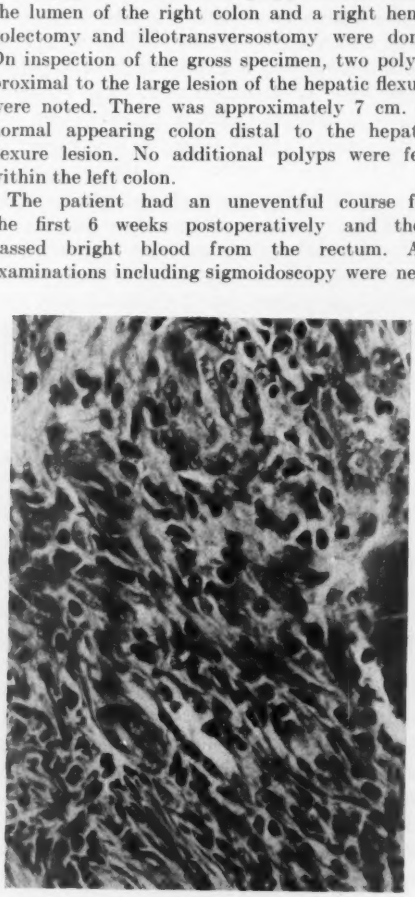

FIG. 1. Case 1, photomicrograph showing invasion of adenocarcinoma, involving muscle of colon.

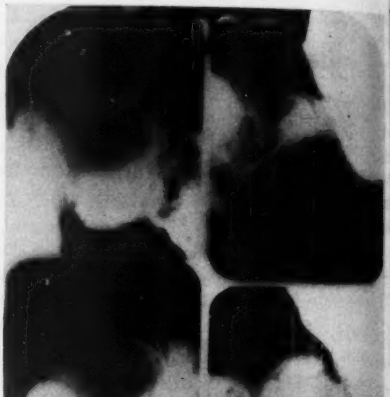

FIG. 2. Case 1, roentgenogram showing deformity of hepatic flexure 6 weeks following resection for carcinoma. Reoperation showed deformity to be due to benign granulomas.

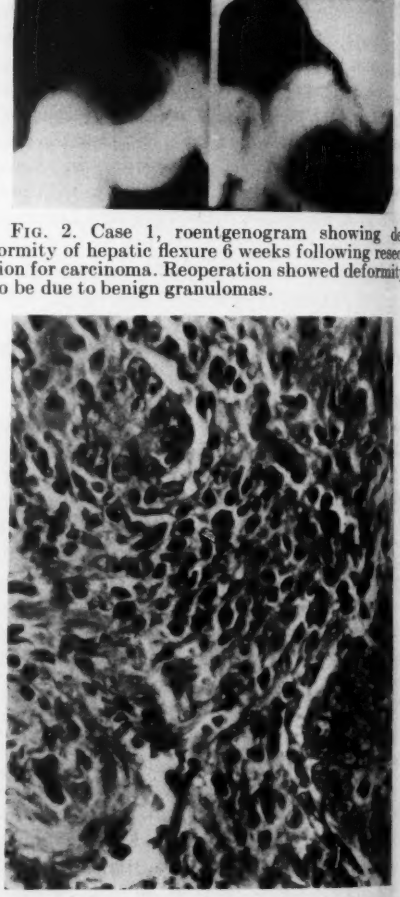

FIG. 3. Case 2, photomicrograph showing adenocarcinoma invading tip of polyp.

With the aid of the proctoscope, the lumen of the bowel was inspected and no additional polyps were found, although an irregular lesion was noted at the line of the midtransverse ileocolostomy. The lesion was resected and an ileotransversostomy was performed. The pathologic diagnosis of the removed lesion was suture granuloma at line of anastomosis (fig. 2). The patient had a normal convalesence and has had no further bleeding.

Case 2. H. H. J., a 67-year-old man, was admitted to Emory University Hospital because of the passing of bright red blood with feces. On proctoscopic examination, his physician had found two small polyps approximately 20 cm. from the anal orifice. Biopsies of these were reported benign. A barium enema demonstrated the possibility of a larger polyp higher up in the sigmoid colon. Upon operation, two pedunculated polyps (0.5 and 1 cm.) were found in the sigmoid colon. The polyps were removed by opening the colon and by local excision. A sigmoidoscope was passed through the colon opening and inserted proximally and distally. No additional lesions were encountered. Pathologic examination of the two polyps revealed adenocarcinomatous changes arising in the tips, without invasion of the stalks (fig. 3).

The patient continued to bleed after removal of the polyps and a repeat barium enema revealed three more polyps. One each was located in the sigmoid, descending, and transverse colon (fig. 4). The patient again was prepared and operated upon 6 months following the original operation. The largest polyp, in the descending colon, was on a stalk and immediate frozen section showed it to be adenocarcinoma. It was felt advisable to do a left colectomy. Three additional small polyps were found in the removed specimen. The patient's course following operation has been uneventful.

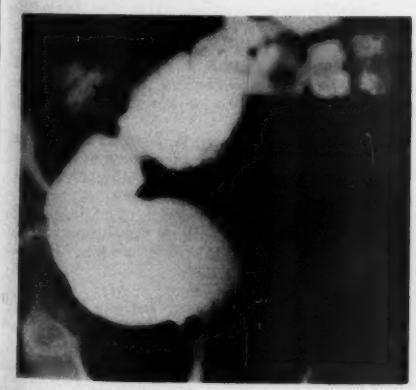

FIG. 4. Case 2, showing typical appearance of polyp which was overlooked as cause for continued bleeding.

FIG. 5. Case 3, roentgenogram showing recurrence following resection of sigmoid colon 1 year before for adenocarcinoma.

Case 3. M. P., a 50-year-old white, single woman, was first seen at Emory University Hospital. She had a 4-year history of loose, bloody, mucous stools. Approximately 1 year earlier she had been operated upon elsewhere and a tumor of the lower colon was removed. Recovery had been uneventful. She began having irregular bowel movements, occasionally loose, and often accompanied by streaks of blood or mucous, 2 months before admission to Emory University Hospital. The patient stated that at the onset of her distress there was a decrease in the caliber of the stool, but recently she had noticed no change. There had been an increase in the passage of flatus and a 2- to 3-pound weight loss. There was no associated abdominal pain (fig 5).

On examination, a questionable mass was palpated in the lower left quadrant. The colon was thought to be palpable in the right lower quadrant as a dilated loop. An exploratory laparotomy was performed and a mass was found in the lower sigmoid colon at the site of the previous resection. A resection of involved area of colon, with an end-to-end anastomosis was performed.

Pathologic examination of the specimen revealed adenocarcinoma of the colon, with extension into the mesentery. The patient had an uneventful postoperative course.

She was seen again at Emory University Hospital 18 months later and stated that she had been well and free from complaints until the night before when she was awakened with rather severe left mid-abdominal pain. A barium enema revealed a well functioning anastomotic site and no evidence of recurrence. Proctoscopic examination was done, with passage of the scope to the distance of 25 cm. The patient was observed for 48 hours and there was no return of pain. It is difficult to state whether this patient has spread of the disease into the abdominal cavity, but it is not believed that there is recurrent disease at the anastomotic site.

SUMMARY

1. Residual symptoms of obstruction or continued bleeding following resection of the large bowel must be viewed with great suspicion of recurrent disease.

2. Normally, during the healing phase following anastomosis of the bowel, radiographic evidences of obstruction may exist. This can be difficult for early distinction from active malignant disease.

3. Necrosis and erosion of the granulomatous change may result in active intestinal hemorrhage and a suspicion of recurrence.

4. Prolonged bleeding should be of great concern since it may occur due to overlooked disease, such as polyps or other malignant lesions, as well as recurrent disease or an inflammatory process.

5. Preventive measures should lessen the possibility of recurrence by careful removal of all disease through an extensive resection, by application of ligatures proximal and distal to the tumor before resection, and by the possible use of anticarcinogenic agents.

6. Even with the utilization of all prophylactic measures, some local recurrences can be expected.

7. If there is a question of recurrent disease, re-exploration is suggested in order to remove remaining disease and to correct any existing obstruction.

8. The likelihood of curing the disease in the late stage by further resection is questionable.

9. Cases are presented demonstrating problems which can be expected following treatment of these conditions.

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REFERENCES

1. Beal, J. M., and Cornell, G. N.: A study of the problem of recurrence of carcinoma at the anastomotic site following resection of the colon for carcinoma. Ann. Surg., *143:* 1, 1956.
2. Cole, W. H.: Measures to combat the menace of cancer. Am. Surgeon, *17:* 660, 1951.
3. Dunning, E. J., Jones, T. E., and Hazard, J. B.: Carcinoma of the rectum: a study of factors influencing survival following combined abdominoperineal resection of the rectum. Ann. Surg., *133:* 166, 1951.
4. Garlock, J. H., and Ginzburg, L.: An appraisal of the operation of anterior resection for carcinoma of rectum and rectosigmoid. Surg. Gynec. & Obst., *90:* 525, 1950.
5. Gilchrist, R. K., and David, V. C.: Prognosis in carcinoma of the bowel. Surg. Gynec. & Obst., *86:* 359, 1948.
6. Goldenberg, I. S., and Hayes, M. A.: Post-obstructive small bowel dyskinesia. Ann. Surg., *147:* 26, 1958.
7. Goligher, J. C., Dukes, C. E., and Bussey, H. J. R.: Local recurrences after sphincter-saving excisions for carcinoma of the rectum and rectosigmoid. Brit. J. Surg., *39:* 199, 1951.
8. Judd, E. S., Jr., and Bellegie, N. J.: Carcinoma of the rectosigmoid and upper part of the rectum. A. M. A. Arch. Surg., *64:* 697, 1952.
9. Lofgren, E. P., Waugh, J. M., and Dockerty, M. B.: Local recurrence of carcinoma after anterior resection of the rectum and sigmoid relationship with the length of normal mucosa excised distal to the lesion. A. M. A. Arch. Surg., *74:* 825, 1957.
10. McGrew, E. A., Laws, J. F., and Cole, W. H.: Free malignant cells in relation to recurrence of carcinoma of the colon. J. A. M. A., *154:* 1251, 1954.
11. Pomeranz, A. A., and Garlock, J. H.: Postoperative recurrence of cancer of colon due to desquamated malignant cells. J. A. M. A. *158:* 1434, 1955.
12. Vink, M.: Local recurrence of cancer in the large bowel; the role of implantation metastases and bowel disinfection. Brit. J. Surg., *41:* 431, 1954.
13. Walt, A. J., and Waugh, J. M.: Certain aspects of recurrence of colonic and rectal cancer; report of three cases. Proc. Staff Meet. Mayo Clin., *31:* 435, 1956.

SIMULTANEOUS OCCURRENCE OF MULTIPLE ARTERIAL EMBOLI IN THE SAME EXTREMITY*

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The increase in the application of direct surgery to cardiovascular lesions has added major sources of arterial emboli such as the walls of heart chambers following valve surgery, dislodged plaques or thrombi during aortic resection or bypass for aneurysm or obliterative disease, and unrecognized thrombi in the lumen of vascular prostheses. The usual sites of origin from the heart and major blood vessels and their multiplicity have been well documented.[1, 4-6, 8, 10, 11] Their simultaneous occurrence in the same extremity has, however, received less emphasis, and may explain the failure of embolectomy to restore adequate circulation to many extremities.

The expansion of the field of vascular surgery has increased the technical abilities of approaching all of the peripheral vessels including the smaller ones for the removal of emboli. This makes the recognition of multiple emboli more important, since most of them can be removed with minimal trauma to the patient and adequate restoration of the circulation to the involved extremity.

Of 15 consecutive patients operated upon by the author during an 18-month period for embolic arterial occlusion, 9 had multiple emboli in the vessels of the same extremity. This report is based on the experiences in these 9 patients.

CLINICAL OBSERVATIONS

The emboli in 7 of the 9 patients originated in the heart, 2 had valvular disease and auricular fibrillation, whereas the others had old or recent myocardial infarctions. Emboli in 1 patient were dislodged in the resection of a large aortic aneurysm and in another while performing a bypass graft from the aorta to the femoral arteries for aortic-iliac stenosis.

Multiple emboli were present in the vessels of the upper extremities of 3 patients (fig. 1). The remainder were in the lower extremities where they were multiple bilaterally in 2 patients and additional single emboli were present in the opposite legs in 2 (figs. 2 and 3). Seven separate emboli were removed from 1 patient (fig. 2*B*).

An accurate clinical diagnosis of acute arterial blockage was possible in all patients. Those admitted for embolic occlusion had excruciating pain, coldness and loss of function of the involved extremity, without pulses distal to the proximal block. The patient with emboli dislodged during the removal of his aneurysm, had good pedal pulses preoperatively. Following insertion of his graft, bounding pulses were restored to the popliteal area but none below. When he awaked both feet and lower legs were cold and painful. Preoperative arteriograms of the patient with emboli dislodged during an aorta-to-femoral artery bypass graft for aortic-iliac stenosis demonstrated patent vessels in the lower legs. Release of the occluding clamps was followed by good pulses in the left leg but none in the right below the superficial femoral artery. Previous knowledge by arteriography that the arteries were patent distal to this point established the presence of acute occlusion.

The patients were given general, spinal, or local anesthesia depending upon their general status and the location of the embolus. The site of exploration was determined by the level of the lowest pulse. The vessels were carefully dissected and manipulation avoided until an occluding clamp was placed distal to the embolus and its tail thrombus. If it was not possible to get below the lower extent through the primary incision, a second one was made at the level of the next major bifurcation. The emboli were removed through longitudinal arteriotomy incisions. When good proximal and distal flow were established, the arteriotomies were closed with a continuous over-and-over suture of fine arterial silk.

Originally, if the retrograde flow appeared adequate and a pulse was restored in the distal vessel, the patients were returned to the recovery room. If the peripheral pulses did not return and

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evidences of acute arterial occlusion were not relieved after 2 to 4 hours, they were returned to the operating room and the distal vessels explored. Later in the series, exploration of the vessels was continued stepwise during the first operative procedure until patency of the entire major vascular tree was established or occlusion by chronic disease demonstrated. Patency in the presence of a weak or questionable pulse was confirmed in 1 patient by exploring the posterior tibial artery at the ankle and detecting a pulse, and in another by arteriography on the operating table.

Most of the emboli were removed through multiple arteriotomy incisions. Retrograde flushing with saline and dilute heparin solution was performed when this could not be done.[2, 3, 7, 9] In the lower extremity flushing was performed from the posterior tibial to the femoral or popliteal and from the popliteal to the femoral arteries, and in the upper extremity from the radial to the brachial or axillary artery. This was not possible in 1 patient with popliteal and posterior tibial artery occlusion, and the emboli were removed by proximal suction through a polyethylene catheter introduced through the popliteal arteriotomy.

The patients with multiple arteriotomies were heparinized systemically and dilute heparin in saline was placed in the distal and isolated arterial segments during the periods of occlusion.

The patients tolerated the operative procedures well and no extremities were lost. One patient died of acute massive myocardial damage and 2 from cerebral emboli during the postoperative period. None of the deaths were related to the operative procedures. The other 6 have good peripheral pulses and no evidence of vascular insufficiency to the extremities.

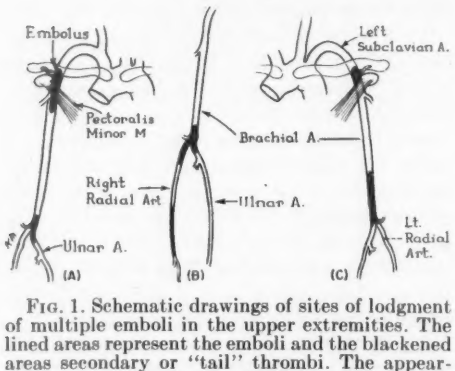

FIG. 1. Schematic drawings of sites of lodgment of multiple emboli in the upper extremities. The lined areas represent the emboli and the blackened areas secondary or "tail" thrombi. The appearance of each is such that distinguishing between an embolus and a thrombus is not difficult. The points of occlusion are: *A*, axillary and terminal brachial; *B*, terminal brachial and radial; and *C*, axillary and midbrachial.

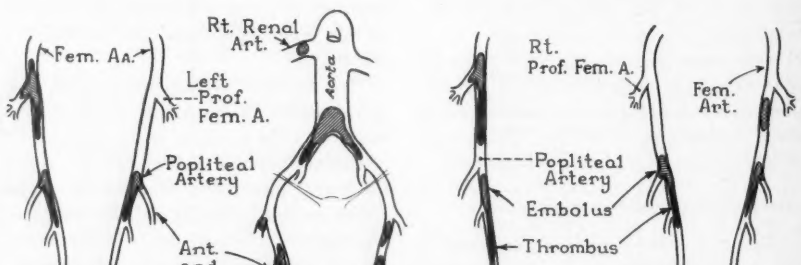

FIG. 2. Schematic drawing of sites of multiple occlusion in vessels of lower extremity by emboli arising in the heart. The emboli and secondary thrombi are depicted as in figure 1. Sites of occlusion are: *A*, right femoral popliteal and posterior tibials and left popliteal; *B*, right renal, bifurcation of aorta; right deep femoral popliteal and posterior tibial; left superficial femoral and popliteal; *C*, right femoral and posterior tibial; and *D*, right popliteal and posterior tibial; left femoral and popliteal.

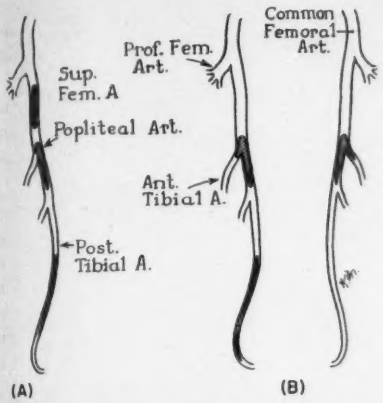

FIG. 3. Schematic drawings of multiple sites of embolic occlusion following aortic surgery. The right superficial femoral popliteal and posterior tibial arteries were occluded by emboli dislodged during insertion of an aorta-to-femoral bypass Teflon graft for aortic-iliac occlusion (*A*). The blockage resulted in thrombosis of the right iliac branch of the graft. These were removed by retrograde flushing from the posterior tibial to the popliteal and popliteal to an opening made in the lower end of the graft. Good pedal pulses were restored. Emboli dislodged during resection and graft replacement of an abdominal aortic aneurysm occluded the right popliteal and posterior tibial and left popliteal arteries (*B*).

DISCUSSION

Multiple emboli in the vessels of the same extremity occurred in 9, or 60 per cent, of 15 consecutive patients. This is a higher incidence than generally reported and actually the series is too small for comparison. The relative frequency in the ones observed is significant, however, and emphasizes the awareness of the possibility in all patients in whom the diagnosis of acute occlusion is made. Manipulation of the vessels was avoided until they were occluded distal to the embolus. It is felt that this eliminated the possibility of the distal emboli resulting from breaking up or dislodging portions of the highest embolus. The emboli either arose multiply or were friable and fragmented in passage or at the point of bombarding the first bifurcation.

The existence of multiple peripheral emboli is best determined by a high index of suspicion. There is such an individual variation in the response to acute arterial occlusion that one may be misled by the clinical findings. All the patients in this series had marked ischemia of the involved extremity but similar findings have been observed in those with a single embolus. Preoperative arteriography was not employed; it adds little to what can be determined clinically and may be harmful, by increasing the arterial spasm or dislodging an embolus.

The surgical approach depends upon the condition of the patient and the point of highest occlusion. General or spinal anesthesia is preferred, but local anesthesia is satisfactory. The entire extremity distal to the highest point of occlusion should be prepared and draped in the operating field to allow palpation of the peripheral pulses and further exploration as necessary.

The patient with a femoral artery embolus is placed in the supine position. The distal common femoral artery and its major branches can then be explored through a longitudinal incision overlying the vessels (fig. 4*A*). Good access to the distal popliteal and tibial arteries can be obtained through a medial incision below the knee retracting the gastrocnemius and soleus muscles. Rotating the foot laterally gives good access to the posterior tibial artery behind the medial malleolus.

The prone position is preferable for patients with embolic occlusion of the popliteal artery. A longitudinal incision below the crease of the

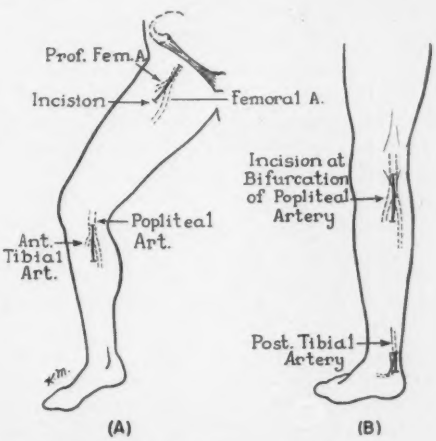

FIG. 4. Schematic drawings of incisions for exposing major vessels of the lower extremities for embolectomy. With patient in the supine position, the femoral popliteal and posterior tibial arteries can be exposed through outlined incisions by lateral rotation and flexion at the knee (*A*). The popliteal and posterior tibial vessels are readily accessible through incisions outlined in "*B*" with patient in the prone position.

popliteal space provides rapid and adequate exposure of the distal popliteal and the origin of the tibial arteries (fig. 4*B*). They heal well and cause little deformity or disability when the popliteal space is not crossed. Access to the posterior tibial artery at the ankle is easily obtained by rotating the foot medially.

It may be difficult to determine the presence of a second embolus after the proximal one is removed. The retrograde flow is diminished but may appear adequate and is difficult to evaluate even by the experienced observer. The most reliable criteria is the failure to establish a pulse in the distal extremity when the proximal block is removed. If it is not definitely known that the vessels are occluded by chronic disease, exploration should be continued until the pulses in the distal extremity are restored. When the pulse at the ankle is questionable, the posterior tibial artery should be exposed. If it is open and not pulsating, one can feel relatively sure that there is a higher occlusion. Arterial spasm may reduce, but is rarely sufficient to prevent, the establishment of a pulsatile flow if the vessels are open. Operating table arteriography may also be employed. Arterial spasm will not prevent the filling of the major vessels to the extremity if they are patent.

Retrograde flushing with saline and dilute heparin solution is a valuable adjunct in removing single or multiple emboli;[2, 3, 7, 9] the anatomy of the arterial tree should be considered in its application. A relatively closed system is necessary to obtain sufficient pressure to dislodge the occluding substance. Patent major arterial branches between the point of flushing and block such as the anterior tibial or peroneal artery may allow sufficient flow to make this impossible. It is occasionally feasible to remove a clot or embolus from the popliteal or femoral artery by flushing from the posterior tibial artery at the ankle to a femoral arteriotomy. It is usually necessary, however, to flush in stages from the popliteal to the femoral and the posterior tibial to the popliteal. The latter is not always possible as was demonstrated in one of our patients in whom the embolus was above the origin of the peroneal artery.

Some emboli and thrombi that have become adherent to the walls of the blood vessels cannot be removed by flushing. Many of these can be removed by repeated suction through a polyethylene catheter inserted into the proximal or distal vessels.[3] Introduction of endarterectomy loops, intraluminal vein strippers, or ureteral catheters of a suitable caliber is often of aid in dislodging the emboli.[3] These methods are not without danger and should not be employed if other methods are successful. Once such is begun it is imperative that a good flow be established since otherwise the trauma produced in the arterial wall will lead to rapid thrombosis.

Regardless of precautionary measures, emboli either single or multiple are always possible during direct surgical approaches to the major blood vessels. Most of these can be managed satifactorily if one is aware of the problem. The status of the peripheral vessels should be determined preoperatively. If the patient has occlusive disease, this should include arteriographic visualization of the major portion of the vessels in the lower legs. If the peripheral vessels are patent and all pulses are not restored when the occluding clamps are removed, an acute occlusion either embolic or thrombotic is probable. The management of both is similar by removal as outlined above. Diligence in this will lead to good results, whereas failure to do so will often result in a miserable failure.

The possibility of multiple emboli increases the importance of early embolectomy. Multiple points of occlusion produce a greater reduction in the flow and increases the rate of progressive thrombosis. This might, particularly in the patient with sclerotic changes in the vessels, eliminate the possibility of successfully restoring the blood flow. If surgery is to be performed at all, it should be done as soon as the diagnosis is established and the patient is operable. Procrastination in trying conservative measures may convert a borderline patient who can have his circulation restored by embolectomy into one that is inoperable.

Success in the management of multiple arterial emboli is dependent upon the development of surgical technique to the point that all of the peripheral vessels can be explored rapidly and with minimal trauma. The continuity of flow in all vessels should be restored. This can be done even in the smaller ones such as the posterior tibial, dorsalis pedis or radial with careful technique utilizing small atraumatic suture.

SUMMARY

Multiple emboli in the vessels of the same extremity occurred in 9 of 15 consecutive patients

on whom embolectomies were performed. Three patients died of causes not related to the operative procedure. The other 6 have good peripheral pulses with no evidence of vascular insufficiency.

The existence of multiple emboli is best determined by a high index of suspicion. The most reliable criterion is the failure to establish a pulsatile flow in the distal extremity when the proximal block is removed.

The successful management of patients with multiple emboli depends upon early diagnosis and surgical treatment, careful technique and persistence until adequate circulation is restored.

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REFERENCES

1. Alergant, C. D.: Sudden simultaneous arterial embolism involving all four limbs. Brit. M. J., *2:* 86, 1954.
2. Crawford, E. S., and DeBakey, M. E.: The retrograde flush procedure in embolectomy and thrombectomy. Surgery, *40:* 737, 1956.
3. Dye, W. S., Olwin, J. H., Javid, H., and Julian, O. C.: Arterial embolectomy. A. M. A. Arch. Surg., *70:* 715, 1955.
4. Eddy, J. P., III, and Congdon, P.: Bilateral non-simultaneous femoral arterial embolic occlusion. Rhode Island M. J., *31:* 669, 1948.
5. Fiddian, J. V.: Simultaneous embolism in both arms. Brit. M. J., *1:* 480, 1949.
6. Haimovici, H.: Peripheral arterial embolism; a study of 330 unselected cases of embolism of the extremities. Angiology, *1:* 20, 1950.
7. Lerman, J., Miller, F. R., and Lund, C. C.: Arterial embolism and embolectomy; report of cases. J. A. M. A., *94:* 1128, 1930.
8. McGarity, W. C., Logan, W. D., Jr., and Cooper, F. W., Jr.: Peripheral arterial emboli. Surg. Gynec. & Obst., *106:* 399, 1958.
9. Shaw, R. S.: A more aggressive approach toward the restoration of blood flow in acute arterial insufficiency. Surg. Gynec. & Obst., *103:* 279, 1956.
10. Scott, H. W., Jr., and Williams, J. M., Jr.: Multiple arterial emboli; 3 successful embolectomies in a case of bacterial endocarditis. Arch. Surg., *58:* 28, 1949.
11. Warren, R., Linton, R. R., and Scannell, J. G.: Arterial embolism. Ann. Surg., *140:* 311, 1954.